IN THE UNITED STATES DISTRICT COURT
FOR THE WESTERN DISTRICT OF VIRGINIA

Civil Action No. 7:14CV00296
NKM

TRUDY ELIANA MUÑOZ RUEDA,

Petitioner,

v.

HAROLD W. CLARKE, Director,
Virginia Department of Corrections,

Respondent.

APPENDIX TO INITIAL PETITION FOR A WRIT OF HABEAS CORPUS SUBJECT TO AMENDMENT

Matthew L. Engle, Virginia Bar No. 46833
THE INNOCENCE PROJECT AT UVA SCHOOL OF LAW
580 Massie Road
Charlottesville, Virginia 22903
(434) 924-2912
(434) 924-4166 (facsimile)

Jonathan P. Sheldon, Virginia Bar No. 66726
SHELDON, FLOOD & HAYWOOD, PLC
10621 Jones Street, Suite 301-A
Fairfax, Virginia 22030
(703) 691-8410
(703) 251-0757 (facsimile)

Article: John Lloyd, et. al., *Biomechanical Evaluation of Head Kinematics During Infant Shaking Versus Pediatric Activities of Daily Living*, Journal of Forensic Biomechanics, Vol. 2, 2011. 152

Article: Ronald H. Uscinski, *Shaken Baby Syndrome: An Odyssey*, Neurol Med Chir (Tokyo) 46, 57-61, 2006. 162

Affidavit of Dr. Ronald Uscinski 168

Affidavit of Guillermo Uriarte 175

Affidavit of Dr. Patrick Barnes 182

Photographs of Muñoz Home 255

EXHIBITS TO MOTION TO DISMISS

Affidavit of Erin Whitmer 260

Affidavit of William E. Hauda, MD 264

Calendar Control Forms 297

EXHIBITS TO REPLY IN SUPPORT OF STATE HABEAS PETITION

Barnes, et al., *Imaging of the Central Nervous System in Suspected or Alleged Nonaccidental Injury, Including the Mimics*, 18 Top Mag Reson Imaging 53 302

Second Affidavit of Dr. Patrick Barnes 325

Photographs of Noah Whitmer 329

Second Affidavit of James Kearney 333

STATE COURT TRANSCRIPTS, ORDERS, AND APPEAL

Transcript of May 23, 2013, Non-evidentiary Hearing 334

Order Dismissing Habeas Petition
(with Transcript of July 24, 2013, attached) 392

Petition for Appeal 414

Order Denying Appeal 455

VIRGINIA

IN THE CIRCUIT COURT FOR THE COUNTY OF FAIRFAX

Case No. _____

TRUDY MUÑOZ RUEDA,
Petitioner,

v.

HAROLD W. CLARKE, Director,
Virginia Department of Corrections,
Respondent.

EXHIBITS TO PETITION FOR A WRIT OF HABEAS CORPUS

Jonathan P. Sheldon, Va. Bar No. 66726
SHELDON & FLOOD, PLC
10621 Jones Street, Suite 301
Fairfax, Virginia 22030
(703) 691-8410
(703) 251-0757 (facsimile)

Matthew L. Engle, Va. Bar No. 46833
THE INNOCENCE PROJECT AT UVA SCHOOL OF LAW
580 Massie Road
Charlottesville, Virginia 22903
(434) 924-2912
(434) 924-4166 (facsimile)

TABLE OF CONTENTS

Exhibit

Noah Whitmer Medical Files Provided by Commonwealth in Discovery (Filed Separately as Sealed CD) ..... A

Affidavit of James R. Kearney ..... B

Affidavit of Renata Ames C

Affidavit of Hernani Ames (Translation by Juliana Ferro) ..... D

Affidavit of Trudy Muñoz Rueda ..... E

Affidavit of Eva Valle (Translation confirmed by Juliana Ferro) ..... F

Affidavit of Michele Shirey ..... G

Affidavit of Rahul Dewan ..... H

Affidavit of Shruti Dewan I

Article: Patrick Barnes, MD & Michael Krasnokutsky, MD, *Imaging of the Central Nervous System in Suspected or Alleged Nonaccidental Injury, Including the Mimics*, Top Magn Reson Imaging, Volume 18, Number 1, February 2007 ..... J

Article: Keith Findley, et. al., *Shaken Baby Syndrome, Abusive Head Trauma, and Actual Innocence: Getting It Right.* ..... K

Article: A. N. Guthkelch, *Infantile Subdural Haematoma and its Relationship to Whiplash Injuries*, British Medical Journal, 1971, 2, 430-431. L

Article: Jan E. Leestma, M.D., M.M., *"Shaken Baby Syndrome": Do Confessions by Alleged Perpetrators Validate the Concept?*, Journal of American Physicians and Surgeons, Vol. 11, No. 1, Spring 2006. ..... M

Article: John Lloyd, et. al., *Biomechanical Evaluation of Head Kinematics During Infant Shaking Versus Pediatric Activities of Daily Living*, Journal of Forensic Biomechanics, Vol. 2, 2011. ..... N

Article: Ronald H. Uscinski, *Shaken Baby Syndrome: An Odyssey*, Neurol Med Chir (Tokyo) 46, 57-61, 2006. ..... O

Affidavit of Dr. Ronald Uscinski ..... P

Affidavit of Guillermo Uriarte ..... Q

Affidavit of Dr. Patrick Barnes ..... R

Photographs of Muñoz Home ..... S

EXHIBIT A

EXHIBIT A – FILED SEPARATELY UNDER SEAL

Medical Records of Noah Whitmer
Provided by the Commonwealth in Pre-trial Discovery

Filed electronically on CD

EXHIBIT B

COMMONWEALTH OF VIRGINIA)
)
COUNTY OF FAIRFAX)

AFFIDAVIT OF JAMES R. KEARNEY

I, James R. Kearney, make the following statement under penalty of perjury:

1. I was one of the lawyers who defended Trudy Muñoz Rueda in her 2010 prosecution for felony child abuse. I was brought into the case by Guillermo Uriarte, Trudy's retained defense lawyer, in order to assist with the examination and cross-examination of medical experts at trial.

2. By experience and training, I am a civil litigation attorney focusing predominantly on personal injury and motor vehicle accident cases. I graduated from Georgetown University Law Center in 1978, and was also admitted to the Virginia Bar in 1978.

3. At the time of Trudy's trial, I had been practicing law for approximately 32 years, but her case was the first criminal felony trial in which I participated. I have a good reputation for litigating in cases involving medical experts, though these have been exclusively civil cases. My experience in criminal defense has been limited to misdemeanor cases. I am not, and have never been, a criminal defense attorney, and was not sufficiently qualified or experienced to handle a felony criminal defense trial.

4. Prior to this case, I knew very little about Mr. Uriarte. I was unaware when I joined the case how inexperienced he was in felony criminal defense matters. Indeed, after I began working with him on this case, at one point I suggested that he step off the case and/or find a more experienced criminal defense attorney to assist him.

5. At some point during the trial, I effectively moved from being second chair to first chair, and I do not feel that I was competent to serve in either role in a felony trial. There were aspects of the trial that I did not understand and was not comfortable with, especially concerning procedural rules. But at the same time, I was also uncomfortable with Mr. Uriarte's apparent lack of experience and felt compelled to assert myself more in the trial.

6. As a defense counsel team, I believe that Mr. Uriarte and I provided ineffective representation to Trudy. Despite our efforts, Mr. Uriarte and I were, at the end of the day, an immigration attorney and a civil litigation attorney, when what Trudy needed was an experienced criminal defense attorney. The bottom line is that Trudy had the wrong two attorneys for the case; I am not a criminal defense

attorney, and I did not realize how inexperienced Mr. Uriarte was until it was too late.

7. I was initially contacted by Mr. Uriarte to participate in the bond reconsideration hearing for Trudy's case and asked to assist him in preparing the expert witnesses at trial. I do not believe that at that time Mr. Uriarte had ever cross-examined an expert witness before. I did not assist in locating or selecting any of the defense experts to be used at trial. Rather, Mr. Uriarte lined up our experts and informed me which experts we would be using.

8. I was shocked at the limited information available to us before trial. Based upon my civil practice, I am accustomed to a much greater amount of discovery. In this case, I was given a CD of crime scene photos from the prosecutor, Mr. Holt, on the first day of trial. Except for a limited number of records, I did not receive discovery from the Commonwealth until mid-December 2009, only a few weeks before our trial began on January 11, 2010. This left us with very little time to work with our experts and prepare our case.

9. I did not receive any reports prepared by the Commonwealth's experts in advance of trial, and did not know what experts the Commonwealth intended to call. Because of this, the only research I was able to do in preparation for cross-examination of the Commonwealth's expert witnesses was what I was able to look up online in the courtroom while they were testifying for the Commonwealth.

10. I was not involved in attempting to secure Dr. Patrick Barnes as an expert witness, who the defense ultimately failed to call as an expert witness on Trudy's behalf. Mr. Uriarte advised me what Dr. Barnes anticipated testifying to, but also told me he was not available for the January 11 trial. We anticipated and relied upon the fact that Dr. Barnes would be able to offer an alternate explanation for the injuries suffered by the child, which did not involve trauma. Such testimony would have provided significant support to the main theory of our defense, which was that there was no abuse of this child. Ultimately, I am uncertain why Dr. Barnes was not secured as an expert at this trial, though I believe it was a scheduling issue.

11. Mr. Uriarte did not discuss with me the possibility of securing a continuance in order to ensure Dr. Barnes' availability, and I never thought of it. Based upon my experience in civil cases, the court will not grant a continuance because the defense needs additional time to prepare its experts. At the time, I did not understand that we had a strong basis for a continuance in light of the fact that the Commonwealth provided over 1000 pages of medical records less than one month before our trial date. It simply never occurred to me, as a civil attorney, that we would have been able to get a continuance of the trial. I recognize now that my understanding was 180 degrees off. We almost certainly could have gotten a continuance, and more time was critical in order to present testimony from our

experts and Dr. Barnes that there were alternate medical explanations for Noah Whitmer's injuries that did not involve trauma.

12. The defense also failed to call Eva Valle, Trudy's sister-in-law, who assisted her with the childcare duties and was at the home the day the child took ill, although she was under subpoena. While I was aware that Ms. Valle was present during the time that Joslyn Waldron, the social worker on the case, interviewed Trudy on April 21, 2009, it did not occur to me that we could use Ms. Valle as a means to impeach Ms. Waldron's testimony. I wish this had occurred to me, as I believe that Waldron's testimony was highly damaging. I believed then and now that it was of the utmost importance to undermine Ms. Waldron's credibility as a witness. If I had known that Ms. Valle had overheard Trudy's interview with Ms. Waldron and could testify that Trudy never admitted wrongdoing despite significant pressure from Ms. Waldron and the police officer to do so, I would have supported putting Ms. Valle on the stand as a witness.

13. I never discussed with Mr. Uriarte the possibility of calling Renata Ames, Trudy's daughter, as a witness. I do not believe that there was ever a strategic decision made not to call Renata as a witness. If I had known that Renata had favorable information about her mother's behavior immediately after Noah became ill and about Noah behaving oddly in the week before he became ill, I would have supported using her as a witness.

15. I don't remember much about Hernani Ames' testimony except that he appeared detached and had somewhat of a flat affect. We did not ask Mr. Ames to testify about the portions of Ms. Waldron's interview of Trudy that he observed on April 21, 2009. This omission was not a strategic decision, but simply an oversight. I believe that impeaching Waldron's testimony was very important and I would have supported presenting testimony from Mr. Ames to discredit her account of that interview if I had known that he had such information.

16. Prior to the trial, I had no experience with Ms. Waldron, the social worker who testified against Trudy, though I had heard from other people that she loves to "play cop," and would testify exactly as she ultimately ended up testifying. She was also the only one in this case who suggested that Trudy had confessed or admitted to guilt in some way. I thought we should have done more with her on cross-examination.

17. Because we never sought a continuance and had so little time to prepare after receiving discovery, and because we were inexperienced in felony criminal trials, Mr. Uriarte and I never settled on a single coherent theory of defense. Instead, we wound up presenting conflicting arguments to the jury. We argued that Noah had not been abused, but also attempted to insinuate that he might have been abused by someone other than Trudy. In my view, the stronger and only really viable defense is that Noah was never abused, because the scientific evidence simply

does not suggest trauma. With more time, we could have presented a much more compelling case explaining his injuries and discrediting Trudy's alleged confession.

18. I continue to believe that Trudy is innocent. Every person I have ever talked to has remarked what a wonderful child care provider Trudy had always been. There was not a shred of evidence in this case that Trudy, in what the Commonwealth suggested was an unwitnessed "fit of rage," ever abused this child. I also believe Trudy to be innocent because, in a physical sense, I do not think she was strong enough to cause those injures, specifically, to cause shearing of the brain inside the skull. I found the fact that the baby had no scratches or bruises on his body, combined with the fact that there were no other injuries to the child's neck, trunk, or extremities, to be very compelling evidence that Trudy did not shake the child.

19. I understand that signing an affidavit is similar to testifying in court. I have carefully reviewed this affidavit before signing it and have been given the opportunity to make any changes to make sure that it accurately reflects my memories about the case.

FURTHER AFFIANT SAYETH NAUGHT.

JAMES R. KEARNEY

Signed and sworn before me this 12th day of November, 2012.

NOTARY PUBLIC

My commission expires: 3/31/2014

Notary registration no.: 7335568

I was commissioned as a Notary as
Vickie Nichole Bradshaw
NOTARY PUBLIC
Commonwealth of Virginia
Reg. #7335568
My Commission Expires 3/31/2014

EXHIBIT C

AFFIDAVIT OF RENATA AMES

I, Renata Ames, make the following statement under penalty of perjury:

1. I am the daughter of Trudy Muñoz Rueda. I was 14-years-old when my mother was arrested, and I remember what happened clearly. I used to live in the United States with my mother and father, but after my mother went to prison I went back to Peru where I live now. Because I grew up in the United States, I am able to speak, read, and write in English.

2. The day that Noah was taken to the hospital was a school day, and I was coming home. Usually my mom would leave the side of the house open because she would be working downstairs in the basement. I went home like any other day and opened the door and went to my room. Usually I go to my room and change my clothes. I was in the house about five minutes when a strange man, a paramedic, walked into my room. This scared me. I followed the paramedic out of the room.

3. When the paramedic and I got to the basement, I saw my mom to the side and Noah on the floor. I went straight to my mom, and the paramedics were taking Noah out. My mom said she was feeding Noah, and he just went limp, so then she gave him CPR. She was really scared. My mom was shaking, so I was just trying to calm her down a little bit.

4. When the paramedics were there, my mom was freaking out, and she was shaking and crying. The other children were also downstairs where the T.V. and games were.

5. A police officer asked my mom what happened, and she told him the same thing that she had told me: she was feeding the baby, and the baby just went limp, so then she gave him CPR. She brought out her binder and showed her certificates and the classes she took for CPR to get her license to show she was authorized to take care of kids. I was out of the room for a moment while the police were interviewing my mom because I got a glass of water to try to calm her down.

6. The police officers interviewed my mom in English and I helped translate. They needed some help translating my mom's words into English because she understands English but has trouble getting her words across in English.

7. That day, when the parents of the other children were coming to pick up their kids, my mom was telling them what happened because the ambulance was still there and that she was sorry that the kids were scared.

8. The next day I came home, and my aunt, Eva Valle-Torres, was there. Shetold me that a social worker and a police officer were interviewing my mom and

that I couldn't be there. I just stayed in my room for a long time, maybe an hour or so.

9. I was often around my mom when she was taking care of other people's children—when I finished my homework, I would help her. I had played with Noah in the weeks before he got sick and had to be taken to the hospital. The week before, I remember he was very cranky. He would cry, and I would give him a toy, and he wouldn't want to play, so I'd give him another toy, and he wouldn't want to play with that one, either. There was nothing I could do to stop him from crying. There was no way for me to play with him. Nothing really calmed him down. He was like this a couple of days before he got sick. Noah's behavior the week before this happened was very unusual for him. Normally, he was a very playful and happy baby. He usually only cried when he was hungry or sleepy.

10. I did not talk to my mom's lawyers about these changes in Noah. I had met Mr. Uriarte before because he was a family friend, but my dad knew him better than I did. My mother's lawyers did not ask me to testify at her trial. If they had asked me to testify, I would have done it. My dad told me I was going to testify, but I think they decided not to have me testify because I might break down and cry.

11. I understand that signing an affidavit is like testifying in court. I have carefully reviewed this affidavit before signing it.

Renata Ames

Signed and sworn before me this 10 day of November 2012, in Lima, Perú (city and country).

EXHIBIT D

COMMONWEALTH OF VIRGINIA)
)
CITY OF CHARLOTTESVILLE)

AFFIDAVIT OF JULIANA FERRO

I, Juliana Ferro, give the following statement under penalty of perjury:

I am a first-year law student at University of Virginia School of Law and a volunteer with the Virginia Innocence Project Student Group. I was born and raised in Colombia, and Spanish is my first language.

Matthew Engle, Legal Director of The Innocence Project Clinic at UVA School of Law, asked me to assist with translation in the case of Trudy Munoz Rueda, because several of the witnesses in that case do not speak English.

Specifically, Mr. Engle asked me to translate an Affidavit signed by Hernani Ames, from English into Spanish, and to send it to Mr. Ames. I have reviewed the Affidavit of Mr. Ames (attached), and attest that the following is an accurate translation of that Affidavit in English, to the best of my ability:

"I, Hernani Ames, make the following statement under penalty of perjury:

1. I am Trudy Munoz Rueda's ex-husband and was married to her at the time of her arrest and trial.

2. I hired Guillermo Uriarte as Trudy's lawyer. I had known him from a long time ago through family relations. I did not contact other lawyers until Mr. Uriarte later hired Jim Kearney to help with the case. Mr. Uriarte told me that he wanted Mr. Kearney's help because Mr. Kearney had experience with accident cases and would be a good lawyer to use.

3. I spoke with Trudy while she was in jail before the trial. I asked Trudy if she had admitted to shaking or jiggling the baby, and she said no. Trudy told me what she had said to the police, but there was a problem with the translation because they took her statements as saying she shook the baby.

4. On April 21, 2009, I came home during Trudy's interview with the social worker and the detective. They had Trudy at the table, and Eva was in the basement. When I arrived, the social worker and the police officer were talking to each other, and they said that Trudy had

already confessed. They said, 'we're going to detain her. We're going to accuse her of mistreatment of a minor.' They said they could arrest Trudy right now or that she could turn herself in to the police later since our daughters were in the house. The social worker and the detective were there for about ten minutes after I arrived. I stayed with Trudy this whole time.

5. Once the two women said Trudy had confessed, I asked Trudy, 'what did you say?' She said I brought the doll to explain how I held the baby. Trudy took the doll and showed me what she had shown the social worker and the detective. Trudy had one hand under the doll's bottom and one behind its back—the way anyone would hold a baby to calm down the baby. Trudy said first the baby was crying, and she had the baby against her chest, and then rocked the baby in front of her.

6. The social worker and the detective were present during Trudy's demonstration with the doll as she showed me. When Trudy demonstrated this motion with the doll, the social worker or the detective said, 'See? That's shaking, that's shaking!' I was surprised when she said 'that's shaking,' because Trudy clearly was not shaking the doll. She was bouncing the doll gently in a soothing way. She was supporting the doll's head with one hand the entire time, and kept the other hand under the doll's bottom. The type of motion that Trudy was demonstrating with the doll was very gentle and could never have injured a baby. I remember the woman making the comment 'See? That's shaking' in English.

7. Eva Valle-Torres stayed in the basement until after the social worker and police officer left. Eva told me, 'I listened to everything' because there was no door to the basement.

8. I had come home on April 21, 2009, because I wanted to be there during the interview, but the social worker and detective came earlier than expected. Trudy said they had been there for about 2-3 hours. I did not argue with hem when they said, 'that's shaking, that's shaking'—I was in shock, partly because I was not expecting them to have been there so early, and partly because I could not believe they were accusing Trudy of hurting Noah.

9. If Mr. Uriarte had asked me about Trudy's interview with the social worker and the detective when I was testifying at her trial, I would have provided the same information that is contained in this affidavit.

10. I am signing this affidavit because Trudy Muñoz Rueda is innocent, and I want to help. I understand that signing an affidavit is like

testifying in court. I have carefully reviewed this affidavit before signing it."

FURTHER AFFIANT SAYETH NAUGHT.

Juliana Ferro

Signed and sworn before me this 12th day of November, 2012.

NOTARY PUBLIC

My commission expires: Aug. 31, 2014

Notary registration no.: 316141

MATTHEW L. ENGLE NOTARY PUBLIC REG # 316141 MY COMMISSION EXPIRES 8/31/2014 COMMONWEALTH OF VIRGINIA

Attachment 1

COMMONWEALTH DE VIRGINIA)
)
CONDADO DE FAIRFAX)

DECLARACIÓN JURADA
(AFIDÁVIT) DE HERNANI AMES

Yo, Hernani Ames, hago las siguientes declaraciones bajo pena de perjurio:

1. Yo soy el ex marido de Trudy Muñoz Rueda y estaba casado con ella en el momento en que fue detenida y durante el juicio.

2. Yo contraté a Guillermo Uriarte como abogado de Trudy. Conocía a Guillermo desde hace mucho tiempo a través de las relaciones familiares. No me comuniqué con otros abogados hasta que el abogado Uriarte después contrató a Jim Kearney para que ayudara con el caso. El abogado Uriarte me dijo que quería la ayuda del Sr. Kearney porque el Sr. Kearney tenía experiencia en casos de accidentes y sería un buen abogado para usar en el caso.

3. Hablé con Trudy mientras estaba en la cárcel antes del juicio. Le pregunté si ella había admitido haber zarandeado o sacudido al bebé, y ella dijo que no. Trudy me contó lo que le había dicho a la policía, pero que hubo un problema con la traducción ya que estos habían interpretado sus declaraciones como si ella hubiera dicho que sacudió al bebé.

4. El 21 de abril del 2009, volví a casa durante la entrevista de Trudy con el la asistente social y la detective. Tenían a Trudy en la mesa, y Eva estaba en el sótano. Cuando llegué, la asistente social y la detective estaban hablando entre ellas, y me dijeron que Trudy había confesado. Dijeron: "Vamos a detenerla. Vamos a acusarla por maltrato a un menor." Dijeron que podían detener a Trudy en ese momento o que ella podía entregarse a la policía más tarde ya que nuestras hijas estaban en la casa. La asistente social y la detective estuvieron en la casa durante unos diez minutos después de que yo llegue. Me quedé con Trudy todo este tiempo.

5. Cuando las dos mujeres dijeron que Trudy había confesado, le pregunté a Trudy, "¿qué has dicho?" Ella dijo que había traído la muñeca para explicar cómo había sostenido al bebé. Trudy tomó la muñeca y me mostró lo que había enseñado a la asistente social y la detective. Trudy tenía una mano detrás de las nalgas de la muñeca y la otra detrás de su espalda – de la misma manera como cualquier persona sostiene a un bebé para calmarlo. Trudy primero dijo que el bebé estaba llorando, y que ella sostuvo al bebé contra su pecho, y luego meció al bebé delante de ella.

6. La asistente social y la detective estaban presentes durante la demostración de Trudy con la muñeca cuando ella me enseño lo que les había mostrado. Cuando Trudy me estaba demostrando este movimiento con la muñeca, la asistente social o la detective dijo: "¿Ves?

Eso es sacudir (zarandear), eso es sacudir (zarandear)!" Me sorprendió cuando me dijo "que eso es sacudir", porque claramente Trudy no estaba sacudiendo la muñeca. Ella estaba meciendo la muñeca de una manera suave y tranquilizadora. Ella sostenía la cabeza de la muñeca con una mano todo el tiempo, y mantenía la otra mano debajo de la las nalgas de la muñeca. El tipo de movimiento que Trudy estaba demostrando con la muñeca era muy suave y nunca hubiera lesionado a un bebé. Recuerdo que la mujer que hizo el comentario "¿Ves? Eso es sacudir (See? That's shacking)" lo dijo en Inglés.

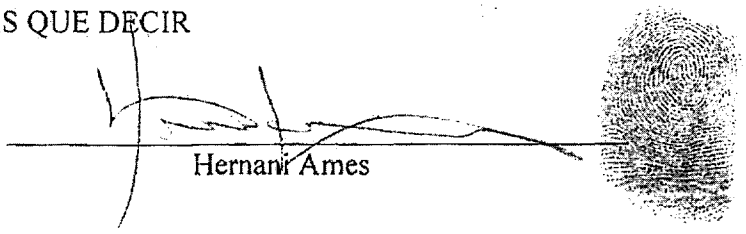
7. Eva Valle-Torres se quedó en el sótano hasta que la asistente social y la detective se fueron. Eva me dijo: "Yo escuché todo" porque no había ninguna puerta hacía el sótano.

8. Yo fui a casa el 21 de abril de 2009 porque quería estar presente durante la entrevista, pero la asistente social y la detective llegaron antes de lo esperado. Trudy dijo que habían estado allí cerca de 2 a 3 horas. No discutí con las mujeres cuando dijeron, "that's shaking, that's shaking"—yo estaba en shock, en parte porque no esperaba que iban a llegar a la casa tan temprano, y en parte porque no podía creer que estaban acusando a Trudy de lastimar Noah.

9. Si el señor Uriarte me hubiese preguntado acerca de la entrevista de Trudy con la asistente social y la detective cuando yo estaba testificando en el juicio, yo hubiera dado la misma información que está contenida en esta declaración jurada (afidávit).

10. Yo estoy firmando esta declaración jurada (afidávit) porque Trudy Muñoz Rueda es inocente, y porque yo quiero ayudar. Entiendo que firmar una declaración jurada (afidávit) es lo mismo que como si estuviese testificando en la corte. He revisado cuidadosamente esta declaración jurada (afidávit) antes de firmarla.

EL DECLARANTE NO TIENE MAS QUE DECIR



Hernani Ames

CERTIFICACION A LA VUELTA

CERTIFICO: QUE LA FIRMA QUE APARECE EN EL ANVERSO DE LA PRESENTE PAGINA, CORRESPONDE A: HERNANI AMES AMEZ, ACREDITO SU IDENTIDAD CON DNI Nº 31676049; DOY FE.

SE EXTIENDE LA PRESENTE CERTIFICACION DE CONFORMIDAD CON EL ARTICULO 108 DE LA LEY DEL NOTARIADO, DECRETO LEGISLATIVO N° 1049, DE LO QUE DOY FE.

LIMA, DOCE DE NOVIEMBRE DEL DOS MIL DOCE.

// LV // 4795// 00062201200004795 //

ALEJANDRO AREVALO MENDOZA
Abogado - Notario de Lima

EXHIBIT E

COMMONWEALTH OF VIRGINIA)
)
COUNTY OF FAIRFAX)

AFFIDAVIT OF TRUDY MUÑOZ RUEDA

I, Trudy Muñoz Rueda, make the following statement under penalty of perjury:

1. I was convicted in *Commonwealth v. Trudy Eliana Muñoz Rueda* of child abuse or neglect and willful or negligent cruelty or injury to a child. I received a sentence of 10 ½ years.

2. After Noah went limp in my arms, and I could not revive him doing CPR, I called 911. The first time I tried to call 911, I dropped the phone. When I finally succeeded, I talked to one of the 911 operators. She coached me to make sure I was doing the chest compressions correctly, and we counted them out loud together. She could tell that I was panicked, and she tried to keep me calm. I kept trying to revive Noah until the ambulance arrived, and then they took over.

3. I told my attorney, Guillermo Uriarte, about my phone call to 911.

4. I did not shake or harm Noah or any other child ever.

⑤ My lawyers never told me they needed more time to get ready for trial. If they had told me that, I would have agreed. I was at home on bond and there was no reason we had to go to trial on January 11, 2010. T.M.

FURTHER AFFIANT SAYETH NAUGHT.

Trudy Muñoz Rueda

Signed and sworn before me this 13th day of November, 2012.

NOTARY PUBLIC

My commission expires: Aug. 31, 2014

Notary registration no.: 316141

MATTHEW L. ENGLE NOTARY PUBLIC REG # 316141 MY COMMISSION EXPIRES 8/31/2014 COMMONWEALTH OF VIRGINIA

EXHIBIT F

COMMONWEALTH OF VIRGINIA)
)
CITY OF CHARLOTTESVILLE)

AFFIDAVIT OF JULIANA FERRO

I, Juliana Ferro, give the following statement under penalty of perjury:

I am a first-year law student at University of Virginia School of Law and a volunteer with the Virginia Innocence Project Student Group. I was born and raised in Colombia, and Spanish is my first language.

Matthew Engle, Legal Director of The Innocence Project Clinic at UVA School of Law, asked me to assist with translation in the case of Trudy Muñoz Rueda, because several of the witnesses in that case do not speak English.

Specifically, Mr. Engle asked me to confirm that Eva Valle's Affidavit (attached) was accurately translated into English. I confirm that the following is an accurate translation of that Affidavit in English, to the best of my ability:

"I, Eva Valle, make the following statement under penalty of perjury:

1. Trudy Muñoz Rueda is my sister-in-law and I worked with her providing child care in her home in Fairfax, Virginia. I usually worked with Trudy in her daycare on weekdays from about 8 AM to 12 PM. Once the babies ate their lunch, I would go home.

2. I planned to testify at Trudy's trial but was not called as a witness. At the time of the trial, I was living in Wisconsin. I had changed my schedule and traveled to Virginia from Wisconsin and stayed there for seven days. Every day of the trial, the attorneys would tell me that I might testify the next day.

3. Trudy's lawyers had told me that I was an important witness because I had worked with Trudy and could say things favorable to her, and I do not understand why they did not call me to testify. They never explained why I was not used as a witness.

5. The week before Noah was taken to the hospital, I worked with Trudy in her daycare because she wanted to be able to give Noah more attention since he was smallest. Trudy cared almost exclusively for Noah and I cared for the other kids in the daycare.

7. On April 20, 2009, I was alone in the daycare for about two to three hours in the morning because Trudy had a doctor's appointment. By the time Noah became ill and had to be taken to the hospital, I was no longer there.

8. Noah was a very calm baby and did not cry. However, the week before April 20, 2009, Noah cried a lot and seemed troubled. One time when I was changing his diaper, I noticed that his poop was abundant and green. I told the police this information when I was interviewed the night of April 20. On that day, my ex husband called me to tell me that the police were at Trudy's house. When I went there, the police asked me questions and I told them about the changes in Noah's poop. I had previously discussed these changes in Noah's poop and his behavior with Trudy. We both noticed that his behavior was strange and wondered if it was because he was teething.

9. On April 21, 2009, I overheard Trudy's interview with a social worker and a police officer. They had asked both Trudy and me to be at Trudy's house to be interviewed on Tuesday. Although we were supposed to be interviewed separately, I could hear Trudy's entire interview. I could not help but listen because the translator was talking loudly in the dining room and I was sitting on the stairs in the basement. The social worker and the police officer took a long time interviewing Trudy and asked her many questions, yet no one asked me any questions. The social worker kept asking Trudy the same thing and accusing her of hurting the baby. I heard them ask Trudy many times what she did to the baby and I heard Trudy say many times that she did not do anything to him.

10. At one point, it seemed like social worker and the police officer gave Trudy something to use to demonstrate her actions but I am not sure because I could not see. Trudy told the women that the baby was crying, so she picked him up. Then the women told her, "show us how you did it." The women were very accusatory and kept pressuring Trudy.

11. Trudy never accepted that she did anything to harm the baby.

12. At the end of the interview, I think that the women asked Trudy if she wanted them to arrest her then in front of her daughters, or later.

13. The women never said anything about whether Trudy needed a lawyer or about recording the interview.

14. Trudy's husband, Hernani, arrived almost at the end of the interview. He never said anything to me about the end of the interview.

15. I have known Trudy for a very long time. She has always been very professional and dedicated to her work. I cannot imagine that she would ever do anything to hurt a child or that she could have hurt Noah accidentally.

16. I am offering this affidavit because I believe Trudy Muñoz Rueda is innocent, and I want to help. I understand that signing an affidavit is like testifying in court. I have carefully reviewed this affidavit before signing it."

FURTHER AFFIANT SAYETH NAUGHT.

Juliana Ferro

Signed and sworn before me this 14th day of November, 2012.

NOTARY PUBLIC

My commission expires: 8/31/14

Notary registration no.: 316141

MATTHEW L. ENGLE NOTARY PUBLIC REG # 316141 MY COMMISSION EXPIRES 8/31/2014 COMMONWEALTH OF VIRGINIA

Attachment 1

STATE OF WISCONSIN)
)
CITY/COUNTY OF Grant)

DECLARACIÓN JURADA (AFIDÁVIT) DE BEATRIZ EVA VALLE - TORO

Yo, Beatriz Eva Valle - Toro, hago las siguientes declaraciones bajo pena de perjurio:

1. Trudy Muñoz Rueda es mi excuñada y yo trabajé con ella en proveer cuidado para niños en su hogar en Fairfax, Virginia. Normalmente yo trabajé con Trudy en la guardería durante la semana desde las 8 de la mañana hasta el mediodía. Después de que los niños almorzaban, yo me iba a mi casa.

2. Yo intenté testificar en el juicio de Trudy pero no se me llamó como una testigo. Cuando empezó el juicio, yo estaba viviendo en Wisconsin. Yo había cambiado mi horario y viajé a Virginia desde Wisconsin. Yo me quedé ahí por siete días. Cada día del juicio, los abogados me dijeron que habria la posibilidad que yo testificaría el próximo día.

3. Los abogados de Trudy me dijeron que yo era una testigo importante porque yo había trabajado con Trudy y podía decir cosas favorables sobre ella, y yo no entiendo porque ellos no me llamaron para testificar. Nunca me explicaron porque no me usaron como una testigo.

5. La semana antes de que Noah fuera al hospital, yo trabajé con Trudy en su guardería porque ella quería darle a Noah mas atención porque el era el mas pequenito. Trudy cuidaba casi exclusivamente a Noah y yo cuidaba a los otros chicos en la guardería.

7. El 20 de abril del 2009, yo estaba sola en la guardería por mas o menos 2 o 3 horas en la mañana porque Trudy tuvo una cita médica. Para cuando Noah se enfermo y fue llevado al hospital, yo ya había salido.

12. Al final de la entrevista, yo pienso que las mujeres preguntaron a Trudy si ella quería que le arresten en este momento en frente de sus hijas, o mas tarde.

13. Las mujeres nunca le dijeron a Trudy nada acerca de que necesitaba un abogado ni tampoco de grabar la entrevista.

14. El marido de Trudy, Hernani, llegó casi al final de la entrevista. El no me dijo nada sobre el final de la entrevista.

15. Yo he conocido a Trudy por muchos años. Ella siempre se ha comportado profesionalmente, y es muy dedicada a su trabajo. Yo no puedo imaginar que ella hiciera cualquiera cosa para hacer daño a un niño ni que ella pudiera hacer daño a Noah por accidente.

16. Ofrezco esta declaración jurada (afidávit) porque creo que Trudy Muñoz Rueda es inocente, y quiero ayudar. Entiendo que firmar una declaración jurada tiene el mismo valor que como si estuviera testificando en la corte. Yo he repasado cuidadosamente esta declaración antes de firmarla.

EL DECLARANTE NO TIENE MAS QUE DECIR.

Evavalle.

Beatriz Eva Valle - Toro

Firmado y atestado ante mí el día 13th de November, 2012.

Nancy A. Grayson

NOTARIO PUBLICO

My commission expires: 8-31-14

Notary registration no.: _____

EXHIBIT G

COMMONWEALTH OF VIRGINIA)
)
COUNTY OF FAIRFAX)

AFFIDAVIT OF MICHELE SHIREY

I, Michele Shirey, make the following statement under penalty of perjury:

1. Trudy Muñoz Rueda was a daycare provider for my two children. Both of my children, Colin and Caleb, started going to Trudy's daycare when they were about three months old and stayed until they were almost two years old.

3. I recommended Trudy as a daycare provider to Noah Whitmer's mom because Trudy gave my contact information to her as a reference. Trudy used me as a reference because I always had good things to say about her. I had no further contact with Mrs. Whitmer, apart from seeing her once at Trudy's house when I went to pick up some things.

4. When Trudy's lawyer contacted me, I agreed to be a character witness because I believe in Trudy and know that she did not do what they accused her of doing. Before I was asked to be a character witness, I was never interviewed by Trudy's lawyers.

5. Trudy's lawyer contacted me about being a character witness about a week before the trial. I was a little stressed because I was given such short notice and had to request a substitute teacher. I had to e-mail the lawyer to find out when and where the trial was being held.

6. Trudy's lawyers told me that they would ask me how long my children had been with Trudy and what I thought about her, but they did not tell me otherwise what questions they would be asking me or prepare me for cross-examination.

7. I followed both Trudy's and Noah's websites because I could not believe what was happening. Trudy was so gentle with my

children that I could not believe she had done anything that could have harmed Noah. As a result, I wanted to advocate for her.

8. I observed Trudy caring for my children because I would sometimes stay and play for about thirty minutes at the end of the day when I would pick my children up. I would stay to play about thirty minutes every other week. I was a very protective parent. I looked at approximately thirty other places to make sure Trudy was the person I wanted as a daycare provider. Trudy was so gentle with my sons. She was never stressed. I never heard her raise her voice. Trudy would take pictures of my boys looking very happy. I was a very protective mother, and Trudy always made me feel very comfortable about my sons' safety and happiness. Colin and Caleb just loved her.

9. I understand that signing an affidavit is like testifying in court. I have carefully reviewed this affidavit before signing it and have been given the opportunity to make any necessary changes.

FURTHER AFFIANT SAYETH NAUGHT.

Michele Shirey

Michele Shirey

Signed and sworn before me this 13 day of November, 2012.

NOTARY PUBLIC

My commission expires: _____

Notary registration no.: _____

Jonathan P. Sheldon
NOTARY PUBLIC
Commonwealth of Virginia
Reg. #318906
My Commission Expires 9/30/2014

EXHIBIT H

COMMONWEALTH OF VIRGINIA)
)
COUNTY OF FAIRFAX)

AFFIDAVIT OF RAHUL DEWAN

I, Rahul Dewan, make the following statement under penalty of perjury:

1. I am the father of Sia Dewan, a child who was under the care of Trudy Muñoz Rueda for approximately eight or nine months. My daughter Sia was under Trudy's care on April 20, 2009, the day that Noah Whitmer, became ill and had to be rushed to the hospital.

2. My wife and I selected Trudy as a day care provider for our daughter Sia based on a referral from one of my wife's friends. We considered many other day care providers before deciding on Trudy.

3. I was always satisfied with Trudy's services as a day care provider. I never had any concerns about leaving our daughter Sia with her, and found Trudy to be a very sweet lady.

4. Both my wife and I were very surprised to hear about the allegations against Trudy. Based upon my interactions with her, it was unbelievable to me then, and is still unbelievable now, that Trudy would have done anything that could have harmed a child in her care.

5. I spoke to Trudy's lawyer once on the phone. The conversation was very general and merely sought to establish how I knew Trudy and what my relationship with her was like. I spoke to Trudy's lawyer only that one time, and was not asked to testify at her trial.

6. I was willing at the time to testify in Trudy's defense, and would have been glad to serve as a character witness for her. Even still, I would be willing to testify or speak on her behalf.

7. I am offering this affidavit because I believe that Trudy is innocent, and I want to help. I understand that signing an affidavit is like

testifying in court. I have carefully reviewed this affidavit before signing it.

FURTHER AFFIANT SAYETH NAUGHT.

RAHUL DEWAN

Signed and sworn before me this 13th day of Nov, 2012.

NOTARY PUBLIC

My commission expires: 5/31/15

Notary registration no.: 7502781

EXHIBIT I

COMMONWEALTH OF VIRGINIA)
)
COUNTY OF FAIRFAX)

AFFIDAVIT OF SHRUTI DEWAN

I, Shruti Dewan, make the following statement under penalty of perjury:

1. I am the mother of Sia Dewan, a child who was under the care of Trudy Muñoz Rueda for approximately eight or nine months. My daughter Sia was under Trudy's care on April 20, 2009, the day that Noah Whitmer, became ill and had to be rushed to the hospital.

2. My husband and I selected Trudy as a day care provider for our daughter Sia based on a referral from one of my wife's friends. I personally interviewed over twenty other day care providers before deciding on Trudy. When I met her I knew she was the one to care for our young child because she was so soft spoken, gentle, and her house was so clean.

3. I was always very satisfied with Trudy's services as a day care provider. I never had any concerns about leaving our daughter Sia with her, and found Trudy to be the sweetest person ever. I cannot imagine even in my dreams that Trudy would hurt a child.

4. Both my husband and I were very surprised to hear about the allegations against Trudy. Based upon my interactions with her, it was unbelievable to me then, and is still unbelievable now, that Trudy would have done anything that could have harmed a child in her care.

5. On the day that Noah went to the hospital I picked up Sia from Trudy's home. I was not called, but came at the regular pick-up time. I remember speaking with Trudy about what happened. Because of her poor English she struggled to tell me that a baby had a problem and went to the hospital. She was clearly stunned and upset.

5. I emailed several times with Trudy's lawyer. The topic was very general and merely sought to establish how I knew Trudy and what

my relationship with her was like. I was not asked to testify at her trial.

6. I was willing at the time to testify in Trudy's defense, and would have been glad to serve as a character witness for her. Even still, I would be willing to testify or speak on her behalf.

7. I am offering this affidavit because I believe that Trudy is innocent, and I want to help. I understand that signing an affidavit is like testifying in court. I have carefully reviewed this affidavit before signing it.

FURTHER AFFIANT SAYETH NAUGHT.

Dewan
SHRUTI DEWAN

Signed and sworn before me this 13th day of November, 2012.

NOTARY PUBLIC

My commission expires: 5/31/15

Notary registration no.: 7502781

EXHIBIT J

Imaging of the Central Nervous System in Suspected or Alleged Nonaccidental Injury, Including the Mimics

Patrick D. Barnes, MD and Michael Krasnokutsky, MD

Abstract: Because of the widely acknowledged controversy in nonaccidental injury, the radiologist involved in such cases must be thoroughly familiar with the imaging, clinical, surgical, pathological, biomechanical, and forensic literature from all perspectives and with the principles of evidence-based medicine. Children with suspected nonaccidental injury versus accidental injury must not only receive protective evaluation but also require a timely and complete clinical and imaging workup to evaluate pattern of injury and timing issues and to consider the mimics of abuse. All imaging findings must be correlated with clinical findings (including current and past medical record) and with laboratory and pathological findings (eg, surgical, autopsy). The medical and imaging evidence, particularly when there is only central nervous system injury, cannot reliably diagnose *intentional* injury. Only the child protection investigation may provide the basis for *inflicted* injury in the context of *supportive* medical, imaging, biomechanical, or pathological findings.

Key Words: child abuse, computed tomography, magnetic resonance imaging, nonaccidental injury, nonaccidental trauma

(*Top Magn Reson Imaging* 2007;18:53–74)

Traumatic central nervous system (CNS) injury is reportedly the leading cause of childhood morbidity and mortality in the United States, resulting in about 100,000 emergencies annually and half the deaths from infancy through puberty.[1–5] The major causes are accidental injuries (AIs) and include falls, vehicular accidents, and recreational mishaps. However, nonaccidental, inflicted, or intentional trauma is said to be a frequent cause, with peak incidence at the age of about 6 months and accounting for about 80% of the deaths from traumatic brain injury in children younger than 2 years. Nonaccidental injury (NAI)—or nonaccidental trauma (NAT)—is the more recent terminology applied to the traditional labels *child abuse*, *battered child syndrome*, and *shaken baby syndrome* (SBS).[4,5] A modern restatement of the definition of SBS is that it represents a form of physical NAI to infants characterized by "the triad" of (1) subdural hemorrhage (SDH), (2) retinal hemorrhage (RH), and (3) encephalopathy (ie, diffuse axonal injury [DAI]) occurring in the context of inappropriate or inconsistent history and commonly accompanied by other apparently inflicted injuries.[6] The short-term life-threatening presentations and long-term outcomes have become a major concern in health care, dating back to the original reports of Kempe,[7] Caffey,[8] and Silverman.[9] Later reports on the incidence rate of CNS trauma in alleged NAI estimate a range of 7% to 19%.[4,5]

However, a number of reports from multiple disciplines have challenged the evidence base (ie, quality of evidence [QOE] analysis) for NAI/SBS as the cause in all cases of the triad.[4,5,10] Such reports indicate that the triad may also be observed in AI (including those associated with short falls, lucid interval, and rehemorrhage) and in nontraumatic or medical conditions. These are the "mimics" of NAI that often present as acute life-threatening events (ALTE). This includes hypoxia-ischemia (eg, apnea, choking, respiratory or cardiac arrest), ischemic injury (arterial vs venous occlusive disease), seizures, infectious or postinfectious conditions, coagulopathy, fluid-electrolyte derangement, and metabolic or connective tissue disorders. Many cases seem multifactorial and involve a combination or sequence of contributing events or conditions.[4,5,10] For example, an infant is dropped and experiences a head impact with delayed seizure, choking spell, or apnea, and then undergoes a series of prolonged or difficult resuscitations, including problematic airway intubation with subsequent hypoxic-ischemic brain injury. Another example is a young child with ongoing infectious illness, fluid-electrolyte imbalance, and coagulopathy, and then experiences seizures, respiratory arrest, and resuscitation with hypoxic-ischemic injury.

Often, the imaging findings are neither characteristic of nor specific for NAI. Because of the widely acknowledged controversy in NAI, the radiologist involved in such cases must be thoroughly familiar with the imaging, clinical, surgical, pathological, biomechanical, and forensic literature from all perspectives and with the principles of evidence-based medicine (EBM).[4,5,10] Children with suspected NAI versus AI must not only receive protective evaluation but also require a timely and complete clinical and imaging workup to evaluate the pattern of injury and timing issues and to consider the mimics of abuse.[4,5,10] All imaging findings must be correlated with clinical findings (including current and past medical record) and with laboratory and pathological findings (eg, surgical, autopsy). The medical and imaging evidence, particularly when there is only CNS injury, cannot reliably diagnose *intentional* injury. Only the child protection investigation may provide the basis for *inflicted* injury in the context of *supportive* medical, imaging, biomechanical, or pathological findings.[4,5,10]

From the Stanford University Medical Center, Stanford, CA.
Reprints: Patrick D. Barnes, MD, Departments of Radiology, Pediatric MRI and CT, Room 0511, Lucille Packard Children's Hospital, 725 Welch Road, Palo Alto, CA 94304 (e-mail: pbarnes@stanford.edu).

MECHANISMS AND MANIFESTATIONS OF TRAUMATIC CNS INJURY

The spectrum of CNS injury associated with trauma (AI or NAI) has been classified into primary versus secondary, focal versus diffuse, and acute versus chronic categories.[4,5,10,11] The primary injury is immediate, irreversible, and is the direct result of the initial traumatic force (eg, contusion, shear injury). Secondary injury denotes the reactive phenomena that arise from or are associated with the primary injury (eg, swelling, hypoxia-ischemia, herniation). Direct contact or impact phenomena produce localized cranial distortion or deformation and thus produce *focal* injury (eg, fracture [Fx], contusion, epidural hematoma [EDH]). Accidental injury is said to be typically associated with this mechanism and result (Fig. 1). Although reported also in cases of NAI, it has been stated that impact injury, with the exception of EDH, is usually not life threatening.

It is *indirect* trauma (ie, independent of skull deformation) that has been considered responsible for the most severe CNS injury in SBS/NAI.[4,5,10–13] Inertial loading accompanying sudden angular acceleration/deceleration of the head on the neck (as with shaking) produces shear strain deformation and disruption at tissue interfaces, therefore *diffusing* the injury (Fig. 2). The young infant is said to be particularly vulnerable because of weak neck muscles, a relatively large head, and an immature brain. It is the shaking mechanism that is traditionally postulated to result in the triad, including primary traumatic injury (ie, SDH, RH, and DAI), with or without the secondary injury pattern (ie, edema, swelling, hypoxia-ischemia, herniation). Reportedly, such patterns are associated with the most severe and fatal CNS injuries and are readily demonstrated by means of neuroimaging, surgical neuropathology, and postmortem neuropathology.[4,5,10–13]

On a medical forensic basis, it is further stipulated that (1) retinal hemorrhages of a particular pattern are diagnostic of SBS/NAI, (2) such CNS injury on an accidental basis can only be associated with a massive force equivalent to a motor vehicle accident or a fall from a 2-story building, (3) such injury is immediately symptomatic and cannot be followed by a lucid interval, and (4) changing symptoms in a child with previous head injury is caused by newly inflicted injury and not just a *rebleed*. Using this reasoning, the last caretaker is automatically guilty of abusive injury, especially if not witnessed by an independent observer.[4,5,10–13]

The range of acute primary and secondary CNS injury reported to occur with NAI significantly overlaps that of AI.[4,5,10,11] This includes multiple or complex cranial fractures, acute interhemispheric SDH (Fig. 2), acute-hyperacute convexity SDH, multiple contusions, shear injury (DAI, white matter tears), brain swelling, edema, and hypoxia-ischemia (Fig. 2). The range of chronic CNS injury includes chronic SDH, communicating hydrocephalus, atrophy, or encephalomalacia. The combination of acute and chronic findings suggests more than 1 traumatic event. Imaging evidence of CNS injury may occur with or without other clinical findings of trauma (eg, bruising) or other traditionally *higher-specificity* imaging findings associated with violent shaking (eg, metaphyseal, rib, or other typical skeletal injuries).[4,5,10] Therefore, clinical and imaging findings of injury disproportional to the history, and injuries of differing age, have become 2 of the key diagnostic criteria indicating the *probability* of NAI/SBS, particularly when encountered in the premobile, young infant.[4,5,10] Such clinical and imaging findings have traditionally formed the basis from which health professionals, including radiologists, have provided a medical diagnosis and offered expert testimony that such *forensic* findings are *proof* of NAI/SBS.[10]

CONTROVERSY

Fundamental difficulties persist in formulating a *medical* diagnosis or *forensic* determination of NAI/SBS on the basis of a causative event (ie, shaking) that is inferred from clinical, radiological, and/or pathological findings in the often *subjective* context of (1) an unwitnessed event, (2) a *noncredible* history, or (3) an admission or confession.[4,5,10] This problem is further confounded by the lack of consistent and reliable criteria for the diagnosis of NAI/SBS, and that the vast body of literature on child abuse is comprised of

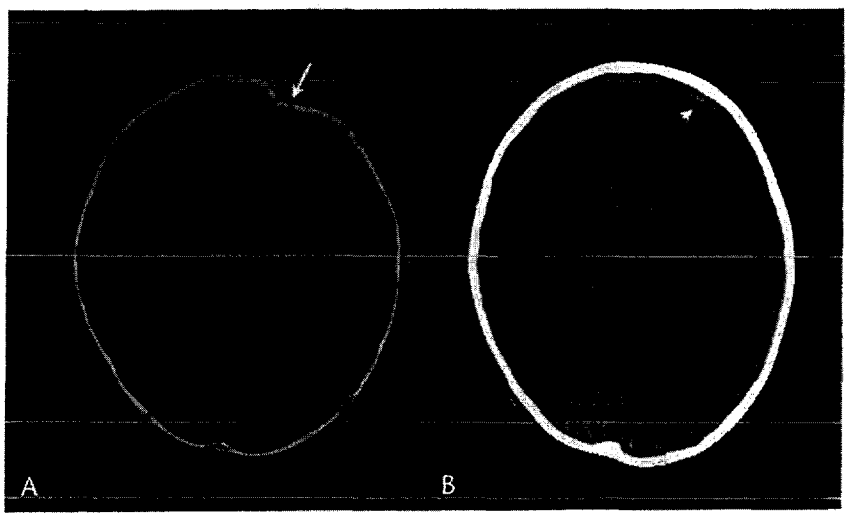


FIGURE 1. Images obtained from a 22-month-old female motor vehicle accident victim with depressed left-side frontal skull fracture (A, arrow), overlying scalp swelling, and a small, high-density epidural hematoma (B, arrowhead).

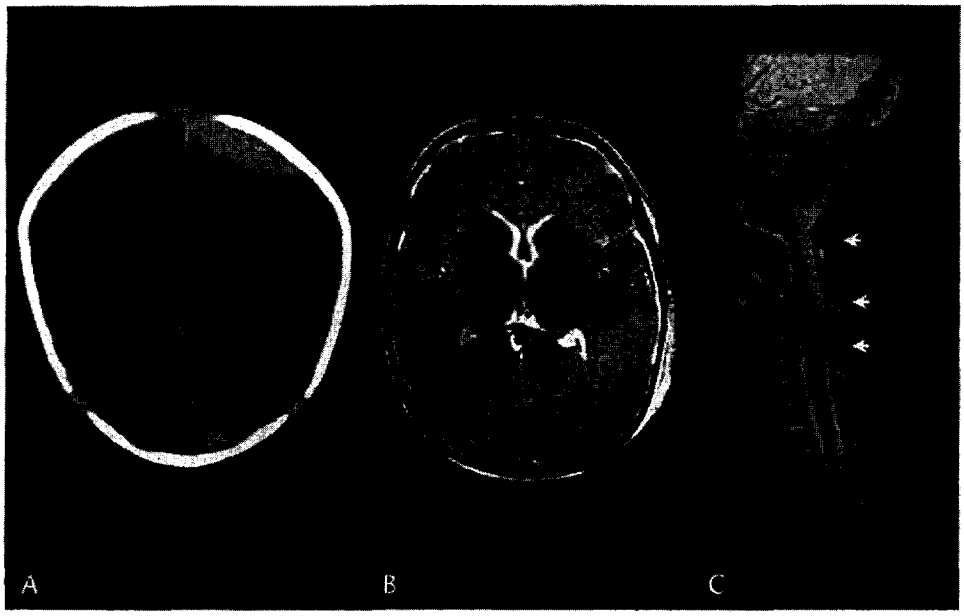


FIGURE 2. Images obtained from a 25-day-old female neonate with history of drop and RH (alleged NAI). A, Axial CT image shows high-density left-side frontal SDH (surgically drained before MRI), bilateral cerebral low densities with decreased gray-white matter differentiation (hypoxia-ischemia?), and interhemispheric high-density hemorrhage. B, Axial T2 MRI scan shows bilateral cerebral cortical and subcortical T2 high intensities plus interhemispheric T2 low intensities. C, Sagittal STIR cervical spine MRI scan shows posterior ligamentous high intensities (arrows) but no definite cord injury (NAI? SCIWORA?).

anecdotal case series, case reports, reviews, opinions, and position papers.[10,14] Furthermore, many reports include cases having impact injury that not only raises doubt regarding the *shaking-only* mechanism but also questions that this injury is always NAI based on a *shaken-impact* mechanism. From the perspective of EBM, QOE ratings for SBS/NAI diagnostic criteria reveal that few published reports merit a rating above class IV (ie, any design where test is not applied in blinded evaluation, or evidence provided by expert opinion alone or in a descriptive case series without controls).[10,14] The inclusion criteria provided in many reports often seem arbitrary, such as *suspected abuse*, *presumed abuse*, *likely abuse*, and *indeterminate*.[15,16] Furthermore, the diagnostic criteria often seem to follow *circular logic* (ie, SBS = SDH + RH [inclusion criteria], therefore SDH + RH = SBS [conclusion]). Such low QOE ratings hardly earn a EBM diagnostic recommendation level of *optional*, much less as a *guideline* or a *standard*.[10,14] This has traditionally been true of the neuroimaging literature, the clinical literature that uses neuroimaging, and the forensic pathology literature.[10,17–44]

The most widely reported attempt of a scientific study to test NAI/SBS used a biomechanical approach, measured stresses from shaking versus impact in a doll model, and correlated those stresses with injury thresholds in subhuman primate experiments established in another study.[45–47] Only stresses associated with impact, whether using an unpadded or padded surface, exceeded the injury thresholds that correlated with the pathological spectrum of concussion, SDH, and DAI. The authors concluded that CNS injury in SBS/NAI in its most severe form is usually not caused by shaking alone. These results obviously contradicted many of the original reports that had relied on the "whiplash" mechanism as causative of the triad.[47–49] These authors also concluded that fatal cases of SBS/NAI, unless occurring in children with predisposing factors (eg, subdural hygroma [SDHG], atrophy, etc), are not likely to result from shaking during play, feeding, and swinging, or from more vigorous shaking by a caretaker for discipline. A number of subsequent studies using various biomechanical, animal, and computer models have failed to convincingly invalidate this study, although many contend that there is no adequate model yet designed to properly test shaking versus impact.[50–61] Some of these reports also indicate that shaking alone cannot result in brain injury (ie, the triad) unless there is concomitant neck, cervical spinal column, or cervical spinal cord injury (Fig. 2).[53,54]

A number of past and more recent reports raise serious doubt that abuse is the cause in all cases of infant CNS injury using traditional SBS/NAI diagnostic criteria.[10,14,16,46,49,62–68] This includes reports of skull fracture or acute SDH from accidental simple falls in young infants, such as those associated with wide extracerebral spaces (eg, benign external hydrocephalus, benign extracerebral collections of infancy, SDHGs),[69–83] and fatal pediatric head injuries caused by witnessed, accidental short-distance falls, including those with a lucid interval and RH.[84–102] Recent neuropathologic studies in alleged SBS cases indicate that (1) the cerebral swelling in young infants is more often caused by diffuse axonal injury of hypoxic-ischemic origin rather than traumatic origin (traumatic origin is more appropriately termed *multifocal traumatic axonal or shear injury*); (2) although Fx, SDH (eg, interhemispheric), and RH are commonly present, the usual cause of death was increased intracranial pressure from brain swelling associated with hypoxia-ischemia; and (3) cervical EDH and focal axonal brain stem, cervical cord, and spinal nerve root injuries were characteristically observed in these infants (presumably caused by shaking, although most had impact findings).[103–109] Such upper cervical cord/brainstem injury may result in apnea/respiratory arrest and be responsible for the hypoxic-ischemic brain injury. Additional neuropathologic series have shown that dural hemorrhages are also observed in nontraumatic fetal, neonatal, and infant cases, and that the common denominator is likely a combination of cerebral

venous hypertension and congestion, arterial hypertension, brain swelling, and immaturity with vascular fragility further compromised by hypoxia-ischemia or infection.[107–109] Reports of neurosurgical, neuroradiological, and neuropathologic findings in head trauma, as correlated with biomechanical analyses, indicate that SDH and RH occur with rotational deceleration injuries, whether *accidental* (eg, axis or center of rotation internal to the skull, including those resulting from short-distance falls) or *nonaccidental* (ie, axis of rotation external to the skull [eg, at the craniocervical junction or cervical spinal level]).[50–53] There is no scientific basis to date to indicate how much or how little force is necessary to produce traumatic injury to the developing CNS.

Furthermore, the specificity of RH for child abuse and its dating has also been questioned.[4,5,10,16,49,67,68,73,84,110–113] Such hemorrhages have been reported with a variety of conditions, including AT, resuscitation, increased intracranial pressure, increased venous pressure, subarachnoid hemorrhage (SAH), sepsis, coagulopathy, certain metabolic disorders, systemic hypertension, and other conditions. Furthermore, many cases of RH (and SDH) are confounded by the existence of multiple factors or conditions that often have a synergistic influence on the type and the extent of RH. For example, consider the child who has trauma, hypoxia-ischemia, coagulopathy, and has undergone resuscitation.

IMAGING PROTOCOLS

Proper imaging evaluation includes not only computed tomography (CT) and a radiographic or radionuclide skeletal survey but also magnetic resonance imaging (MRI) and, in some cases, serial imaging.[4,10,114–118] Occasionally, ultrasonography (US) may be useful. The imaging protocols should be designed to evaluate not only NAI versus AI but also the nontraumatic mimics. Computed tomography is the primary modality in acute neurological presentations because of its access, speed (particularly using multidetector technology), and ability to demonstrate abnormalities requiring immediate neurosurgical or medical intervention (eg, an expanding hematoma, brain swelling, impending herniation) (Figs. 1, 2).[4,10,114] Nonenhanced head CT with soft tissue and bone algorithms is performed. Facial and spinal (eg, cervical) CT may also be needed, including reformatting. Three-dimensional computed tomographic reconstructions can be important to evaluate fractures versus developmental variants (eg, accessory sutures, fissures, synchondroses). Computed tomographic angiography (CTA) or computed tomographic venography (CTV) may be helpful to evaluate the cause of hemorrhage (eg, vascular malformation, aneurysm) or infarction (eg, dissection, venous thrombosis). Intravenous contrast-enhanced CT or US with Doppler may be used to separate subarachnoid and subdural compartments by identifying bridging veins within the subarachnoid space; however, MRI is usually needed for more definite evaluation. In addition, in the unstable infant, initial and repeat cranial US (eg, transcranial Doppler) at the bedside may assist in evaluating structural abnormalities and monitoring alterations in cerebral blood flow and intracranial pressure.

Magnetic resonance imaging should be conducted as soon as possible because of its sensitivity and specificity regarding pattern of injury and timing parameters.[4,10,114–118] Brain MRI should include 3 planes and at least T1, T2, fluid-attenuated inversion recovery (FLAIR), gradient-recalled echo (GRE) T2*, and diffusion imaging (diffusion-weighted imaging [DWI]/apparent diffusion coefficient [ADC]) (Fig. 3). Gadolinium-enhanced T1 images should probably be used along with MRA and magnetic resonance venography (MRV).

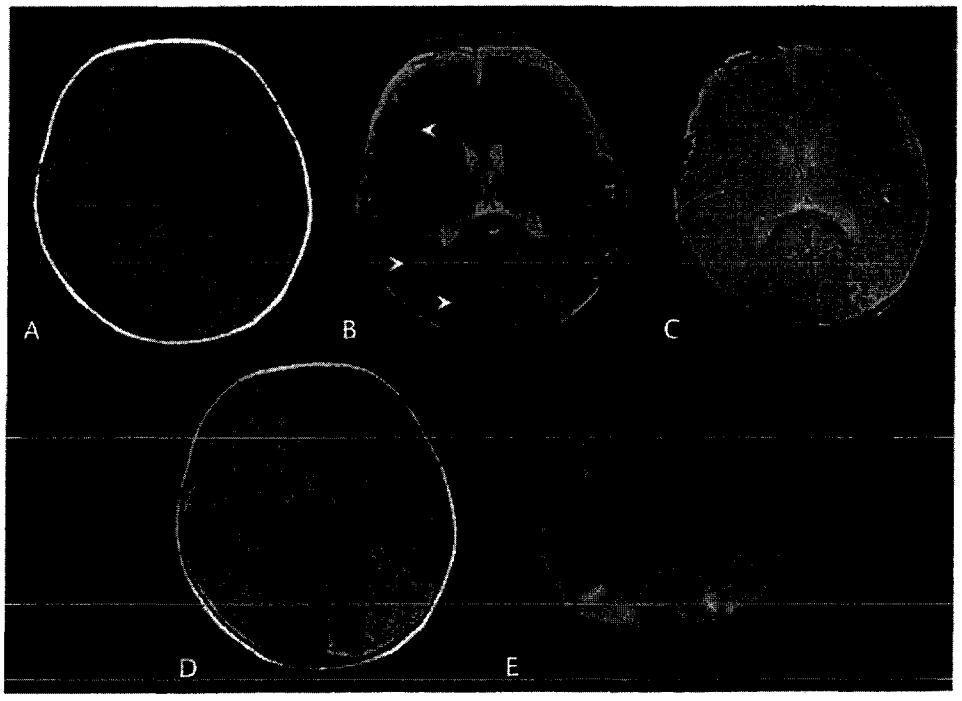


FIGURE 3. Images obtained from an 8-month-old male infant after viral illness, right-side humeral fracture, and RH (alleged NAI). Axial T1 (A), T2 (B), GRE (C), FLAIR (D), and DWI (E) images show bilateral frontal extracerebral CSF-intensity collections with right-side frontal extracerebral hemorrhage that is T1/FLAIR hyperintense and T2/GRE hypointense. Also seen are multifocal cerebral T2/FLAIR hyperintensities (arrowheads) that are DWI hyperintense (shear vs infarction?).

TABLE 1. Magnetic Resonance Imaging of Intracranial Hemorrhage and Thrombosis*

Stage	Biochemical Form	Site	T1 MRI	T2 MRI
Hyperacute (+ edema) (<24 hours)	Fe II oxyHb	Intact RBCs	Iso-low I	High I
Acute (+ edema) (1–3 days)	Fe II deoxyHb	Intact RBCs	Iso-low I	Low I
Early subacute (+ edema) (3–7 days)	Fe III metHb	Intact RBCs	High I	Low I
Late subacute (− edema) (1–2 weeks)	Fe III metHb	Lysed RBCs (extracellular)	High I	High I
Early chronic (− edema) (>2 weeks)	Fe III transferrin	Extracellular	High I	High I
Chronic (cavity)	Fe III ferritin and hemosiderin	Phagocytosis	Iso-low I	Low I

*Modified from Wolpert and Barnes,[119] Kleinman and Barnes,[4] Bradley,[120] and Zuerrer et al.[121]
RBCs indicates red blood cells; I, intensity; plus sign (+), present; minus sign (−), absent; Hb, hemoglobin; Fe II, ferrous; Fe III, Ferric; Iso, isointense.

The cervical spine should also be imaged, along with other levels when indicated, and especially by using short TI inversion recovery (STIR) (Fig. 2). T1- and T2-weighted imaging techniques are necessary for characterizing the nature and timing (whether hyperacute, acute, subacute, or chronic) of hemorrhages and other collections by using established criteria (Table 1). Gradient-recalled echo or other susceptibility-weighted (T2*) techniques is most sensitive for detecting hemorrhage or thromboses that are often not identified on other sequences. However, GRE cannot be used for timing alone because it shows most hemorrhages (new and old) as hypointense (eg, deoxyhemoglobin, intracellular methemoglobin, hemosiderin).[4,10,114] The FLAIR sequence suppresses cerebrospinal fluid (CSF) intensity and allows for a better assessment of brain abnormalities, especially when adjacent to a CSF space or collection. FLAIR is also sensitive (but nonspecific) for subarachnoid space abnormalities, which appear as high intensity (eg, hemorrhage, exudate, inflammatory or neoplastic leptomeningeal infiltration, occlusive vascular slow flow, and hyperoxygenation during sedation or anesthesia). DWI plus ADC can be quickly obtained to show hypoxia-ischemia or vascular occlusive ischemia. Magnetic resonance spectroscopy (MRS) may show a lactate peak. It must be remembered, however, that restricted or reduced diffusion may be observed in other processes, including encephalitis, seizures, or metabolic disorders, and with suppurative collections and some tumors.[4,10,114] Gadolinium-enhanced sequences and MRS can be used to evaluate these other processes. In addition, MRA and MRV are important to evaluate arterial occlusive disease (eg, dissection) or venous thrombosis. The source images should be viewed along with the reprojected images. In some cases of partial occlusion/thrombosis, the abnormality may be more conspicuous on CTA/CTV, especially in infants. For evaluating arterial dissection by means of MRI, an axial fat-suppressed T1 sequence from the aortic arch to the circle of Willis may detect T1-hyperintense hemorrhage or thrombosis (ie, methemoglobin) within the false lumen, especially if the process is in the subacute phase.

INJURY EVALUATION

The range of CNS injury in childhood trauma, whether AI or NAI, often demonstrated by imaging may be categorized according to being primary or secondary (as previously described) and according to specific anatomical involvement, including scalp, cranial, intracranial, vascular, spinal, and head and neck.[2,4,5,10] A thorough analysis of the injury requires a systematic breakdown into injury components for both pattern of injury and timing parameters.

SCALP INJURY

Scalp injuries include hemorrhage, edema, or laceration and may be localized to any layer (SCALP [*s*kin, subcutaneous,

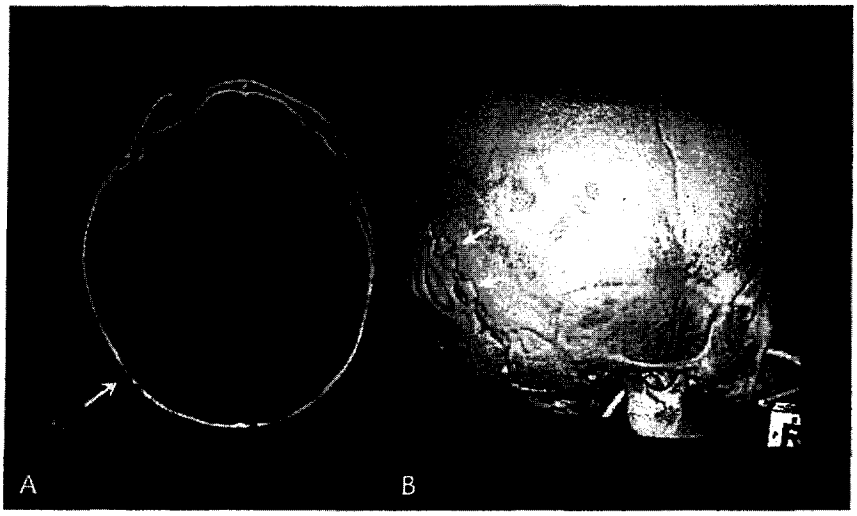


FIGURE 4. Images obtained from a 10-month-old male infant with intrasutural (wormian) bones versus fractures. A, CT image shows right-side parietal cranial defects (arrow). B, Three-dimensional computed tomographic surface reconstruction confirms intrasutural bones (arrows).

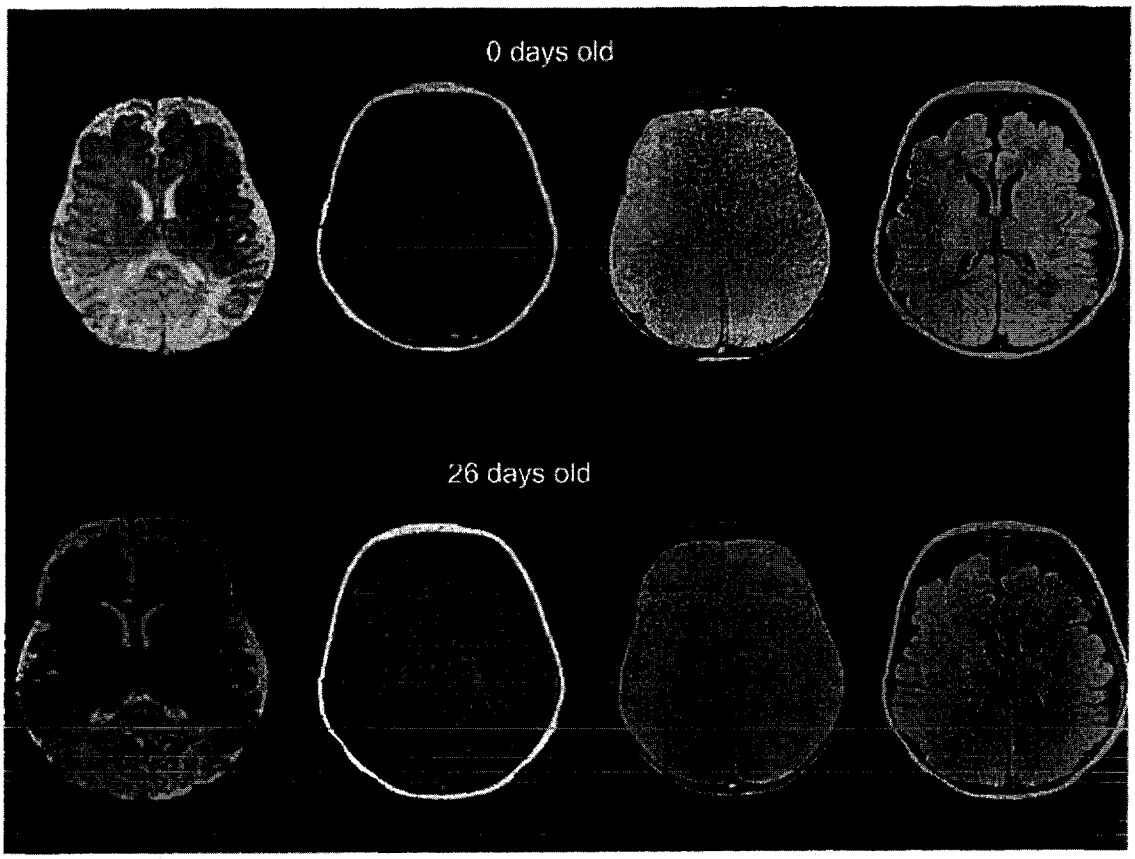


FIGURE 5. Images obtained from an infant with benign extracerebral collections of infancy and spontaneous subdural hemorrhage. Axial T2, T1, GRE, and FLAIR images (left to right) show CSF-intensity frontal subarachnoid collections at birth (top row). At 26 days postnatal age (bottom row), superimposed subdural collections that don't conform to CSF signal are present (courtesy of Veronica J. Rooks, MD, Tripler Army Medical Center, Honolulu HI).

galea *a*poneurotica, *l*oose or subga*l*eal space, *p*eriosteum]).[2,4,5,10] Although CT or MRI may not precisely resolve scalp layers, the site of a collection may be inferred by means of morphological findings (Fig. 1). Subperiosteal collections (eg, cephalohematoma) are usually confined by the sutures. Subcutaneous or subgaleal collections are not as contained, may be more extensive, and can contribute to circulatory compromise. Scalp injuries are difficult to precisely time on imaging studies, unless serial examinations are available; in addition, timing depends on the nature and the number of traumatic events or other factors (eg, circulatory compromise). Unless there is direct vascular injury that results in an acute hematoma, collections or edema may not be identified on early imaging. Scalp injuries may become evident several hours later or on the next day. Nonvisualization of scalp or skull abnormalities on imaging should not be interpreted as absence of impact injury.

SKULL INJURY

The spectrum of cranial injury includes Fx*s* and suture splitting.[2,4,5,10] Fractures may be simple (eg, single, linear, nondisplaced) or complex (eg, bilateral, multiple, diastatic, depressed, or growing [ie, leptomeningeal cyst]). Localized suture splitting may indicate traumatic diastasis where widening occurs as a part of Fx extension. Diffuse or multiple suture widening may indicate increased intracranial pressure from any cause to include edema, expanding collection, or hydrocephalus. Evaluating the skull in neonates, infants, and young children is challenging because Fx may not be distinguished from sutures, synchondroses, or their normal variations. This is particularly difficult in the parietooccipital region and skull base where accessory sutures, fissures, and synchondroses are common. The significance of this distinction is important because the reporting of a skull Fx is *evidence* of trauma (Fig. 1). In such cases, 3-dimensional computed tomography with surface reconstructions may provide clarification (Fig. 4). In general, the morphology of an Fx does not differentiate NAI from AI. Complex or bilateral skull Fx in this age group can arise from a single event under circumstances other than a 2-story fall or a motor vehicle accident. Such examples include a fall or a drop with impact to the skull vertex, impact against more than 1 surface (eg, table, wall, or floor), fall or drop downstairs, and an adult or older child falling with or onto a smaller child. Skull Fx*s* are also difficult to time by using plain films and CT because of the lack of periosteal reaction during healing. A simple skull Fx in an infant may require 6 months for complete healing. In an older child and adult, this may take up to a

year.[2,4,5,10] Intracranial air densities (ie, pneumocephalus) may be related to fracture involving the paranasal sinuses or otomastoid structures, caused by penetrating trauma (eg, open skull fracture), arise from CSF access (eg, lumbar puncture) or vascular access (eg, indwelling catheter), or may be associated with gas-forming infections.

EXTRACEREBRAL COLLECTIONS

The range of intracranial injury includes abnormal fluid collections and brain injury.[2,4,5,10] Abnormal collections may be subarachnoid, intraventricular, subdural, or epidural. These may contain hemorrhage of any age (eg, hyperacute, acute, subacute, chronic, combined), cerebrospinal fluid (CSF [eg, hygroma, hydrocephalus]), protein, exudate, or any combination of elements. On imaging, it may be impossible to specifically define the components or age of a collection (eg, SDHG vs chronic SDH). Subarachnoid and subdural collections may be localized or extensive and occur near the convexities, interhemispheric (along the falx), and along the tentorium. Epidural hemorrhage, whether arterial or venous in origin, tends to be more localized (limited by the periosteal layer of the dura mater along the inner calvarial table) and can cross midline (Fig. 1). Epidural (intradural) hemorrhage may split the leaves of dura and collect within the tentorium or falx. Epidural collections usually appear lentiform. Subdural collections tend to be crescentic and follow the contour of the adjacent cerebrum or cerebellum (Fig. 3). Subarachnoid collections may be less well defined (unless loculated) and extend into cisterns, fissures, or sulci. Occasionally, a collection cannot be determined to be specifically subarachnoid, subdural, or epidural because collections in multiple spaces may be present, owing to membrane layer disruption (Fig. 2). Intraventricular hemorrhage is a rare but reported finding in trauma. It may also be an indicator of associated hypoxia-ischemia, coagulopathy, or venous thrombosis.

Prominent subarachnoid CSF spaces may normally be present in infants (aka benign extracerebral collections [BECC], benign extracerebral subarachnoid spaces, benign external hydrocephalus).[10,79–83,114] These should be of the same density/intensity as CSF on CT and MRI (Fig. 5). This condition predisposes infants to SDH, which may be spontaneous or associated with trauma of any type (Fig. 5). A hemorrhagic collection may continually change or evolve with regard to size, extent, location, and density/intensity characteristics. Cases of rapid resolution and redistribution of acute SDH for a few hours to 1 to 2 days have been reported.[117,122] A tear in the arachnoid may allow SDH washout into the subarachnoid space or CSF dilution of the subdural space. An SDH may also redistribute within the subdural space as a gravity-dependent process (eg, a convexity SDH migrating to the peritentorial and posterior interhemispheric regions)[114,117] (Fig. 6). Subdural hemorrhage migration may lead to misinterpretation of a new hemorrhage. The distribution or migration of the sediment portion of a hemorrhage with blood levels (ie, hematocrit effect) may

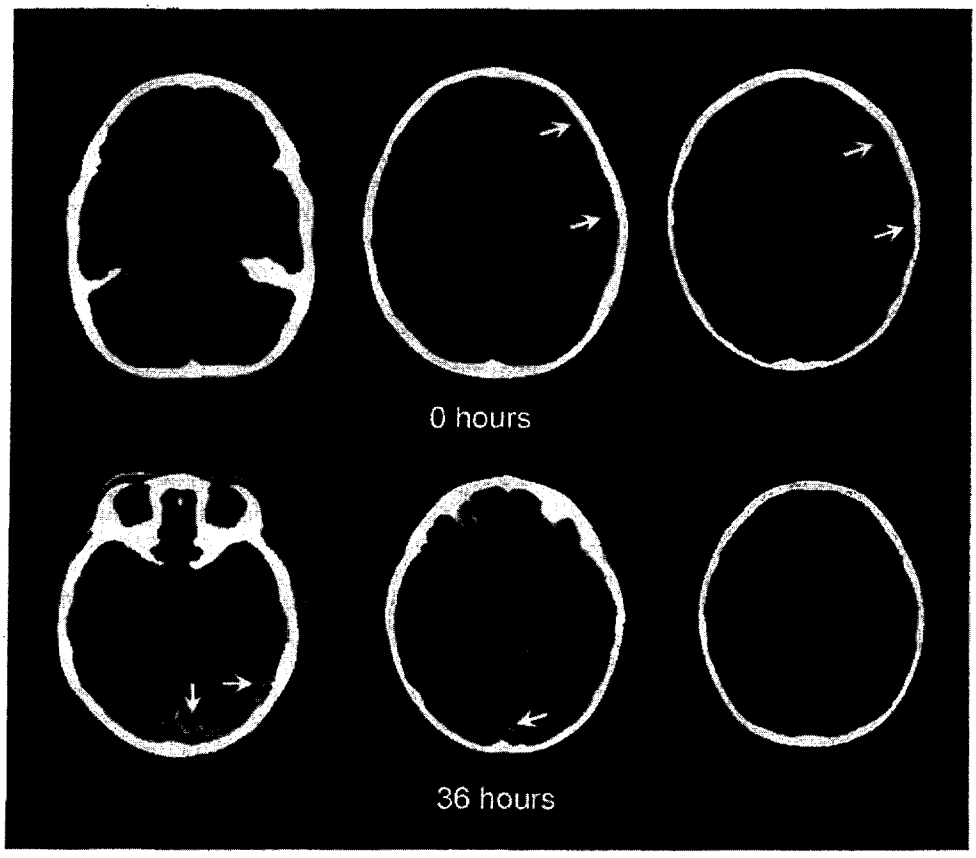


FIGURE 6. Images obtained from a 9-month-old female infant who had accidental trauma from left-side frontal impact. Computed tomographic images at presentation (top row) show left-side frontotemporal-convexity high-density subdural hemorrhage (arrows). Computed tomographic images obtained after 36 hours in the hospital (bottom row) show redistribution of the high-density hemorrhage to the peritentorial region and posterior interhemispheric fissure (arrows).

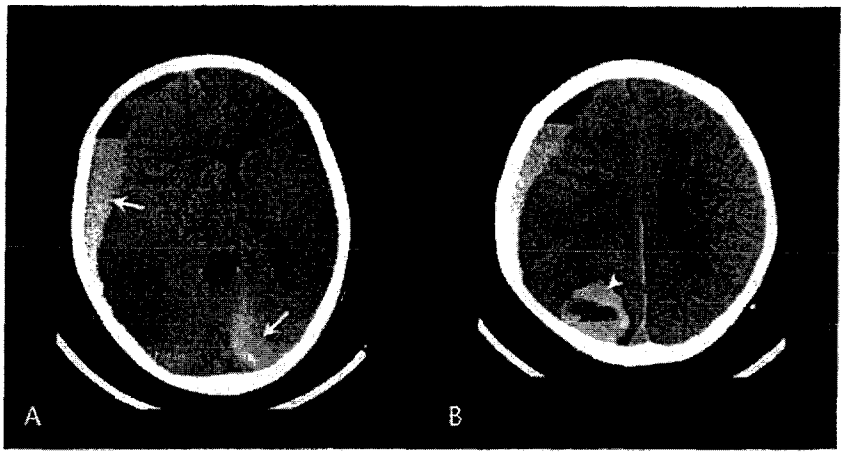


FIGURE 7. Images obtained from a 2-year-old boy with congenital heart disease and ECMO. Axial computed tomographic images show bilateral subdural hematomas (A, arrows) and right-side parietal intracerebral hematoma (B, arrowhead) with low-density over high-density fluid levels.

cause further confusion because the density/intensity differences between the sediment and supernatant may be misinterpreted as hemorrhages (and trauma) of differing age and location (Figs. 7, 8).[117] In addition, more recent reports further substantiate that (1) the interhemispheric SDH may be observed in AI and, therefore, is not specific for NAI; (2) mixed-density SDH also occurs in AI; (3) SDH may occur in BECC either spontaneously or as a result of minor trauma (ie, AI); and (4) rehemorrhage within SDH may occur spontaneously or with minor AI.[10,82,114–118]

BRAIN INJURY

Traumatic brain injury includes contusion, shear injury, hemorrhage, and edema.[2,4,5,10] Contusions represent focal or multifocal impact injury, are usually hemorrhagic, and typically occur in cortical gray matter along brain surfaces that impact skull bone or dura mater (eg, falx, tentorium). The inner table of the immature, infant skull is not as *rough* as in older children and adults. Therefore, sliding contusions of the frontal or temporal lobes along the floor of the anterior or middle cranial fossa, respectively, occur less often. Infant contusions more commonly occur at the primary site of impact (ie, coup injury) or at a secondary, "recoil" site opposite the primary impact (ie, contracoup injury). Shear injury (ie, traumatic axonal injury, white matter tear) is also focal or multifocal and typically occurs at deep gray matter–white matter junctions, along the corpus callosum, and within the brain stem (Fig. 3). They are more often nonhemorrhagic but may become hemorrhagic. In severe cases, shear injuries may appear as gross tears. This type of injury has been previously referred to as *diffuse axonal injury* or DAI. It is more properly termed *multifocal or traumatic axonal injury* because diffuse axonal injury is more characteristic of hypoxic-ischemic injury (Fig. 2).[104–109]

Edema or swelling may be traumatic, hyperemic, hypoxic-ischemic, or related to other factors (eg, seizures, metabolic).[2,4,5,10] Traumatic edema is related to direct traumatic effects such as contusion, shear, or the result of a vascular injury (eg, dissection, herniation) (Figs. 2, 3). Malignant brain edema, a term used for severe cerebral swelling leading to rapid deterioration, may also occur in children with head trauma. The edema may be related to cerebrovascular congestion (ie, hyperemia) as a vasoreactive rather than an autoregulatory phenomenon. There may be rapid or delayed onset.[84–96] Predisposing factors are not well established but likely include a genetic basis. Global hypoxia (eg, apnea, respiratory failure) or ischemia (eg, cardiovascular failure or dissection) is likely a major cause of or contributor to brain edema in the child with head trauma (Fig. 2). Other contributors to edema or swelling include such complicating factors as seizures (eg, status epilepticus), fluid-electrolyte imbalance, other systemic or metabolic derangements (eg, hypoglycemia, hyperglycemia, hyperthermia), or hydrocephalus. The type (eg, cytotoxic, vasogenic, hydrostatic) and pattern of edema tend to conform to the nature and distribution of the causative insult. Traumatic edema is often focal or multifocal (eg, in areas of contusion, shear, or hemorrhage) (Fig. 3). Hyperemic edema is often diffuse and may appear early as accentuated gray-white matter differentiation on CT, then progressing to loss of differentiation (Fig. 2). Hypoxic-ischemic injury, depending on its severity and duration, may have a diffuse appearance acutely with decreased gray-white matter differentiation throughout the cerebrum on CT (eg, white cerebellum sign) and then evolve to a more specific pattern on CT or MRI (eg, border zone or watershed, basal ganglia/thalamic, cerebral white matter necrosis, reversal sign) (Fig. 2).[10,114,123–126] The subacute to chronic sequelae of traumatic brain injury include hydrocephalus, atrophy, encephalomalacia, gliosis, mineralization, and chronic extracerebral collections.

VASCULAR INJURY

Arterial trauma may result in dissection or pseudoaneurysm.[2,4,5,10,123, 127] The vascular injury may be the result of penetrating or nonpenetrating trauma, may be spontaneous, or caused by existing disease (eg, arteriopathy). Internal carotid artery dissection typically involves the cervical or supraclinoid segments. Vertebrobasilar dissection most commonly involves the distal cervical portion of the vertebral artery at the C1-C2 level. Intracranial or multiple dissections may rarely occur. Dissection may result in stenotic, thrombotic, or embolic infarction. Pseudoaneurysms may be associated with hemorrhage. The vascular injury may be initially detected by means of CT and CTA (Fig. 9) or of MRI (eg, DWI, axial fat-suppressed T1 sections of the neck and skull base) with MRA. Catheter

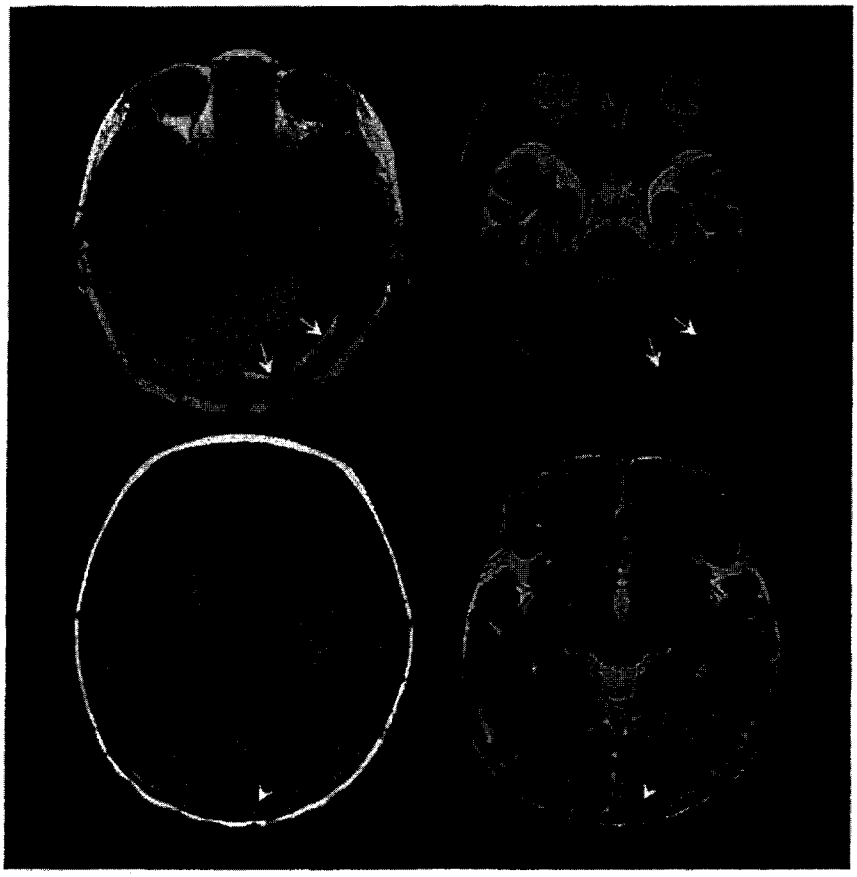


FIGURE 8. Images obtained from a 2-month-old female infant with left-side peritentorial and posterior interhemispheric subdural hemorrhage. Axial MRI images show T1-hyperintense and T2-hypointense sediment along the tentorium (top row, arrows) with T1- and T2-isohyperintense supernatant above (bottom row, arrowheads).

angiography may be necessary for definitive evaluation. Arterial occlusive infarction also occurs with the various types of herniation, in which relatively specific distributions are observed. Dural sinus and venous thrombosis may also occur with trauma (eg, adjacent to fracture, associated or predisposing coagulopathy) or as a mimic of NAI (eg, infection, coagulopathy).[128] Computed tomography may show hyperdensity within the venous system, a focal venous enlargement with associated subarachnoid or subdural hemorrhage, or infarction that is often hemorrhagic. A more definitive diagnosis may be made by means of CTV or of MRI and MRV.

SPINAL INJURY

The spectrum of spinal injury in NAI significantly overlaps that of AI.[2,4,5,10,123] This spectrum differs with age (degree of spinal development) and includes either single or

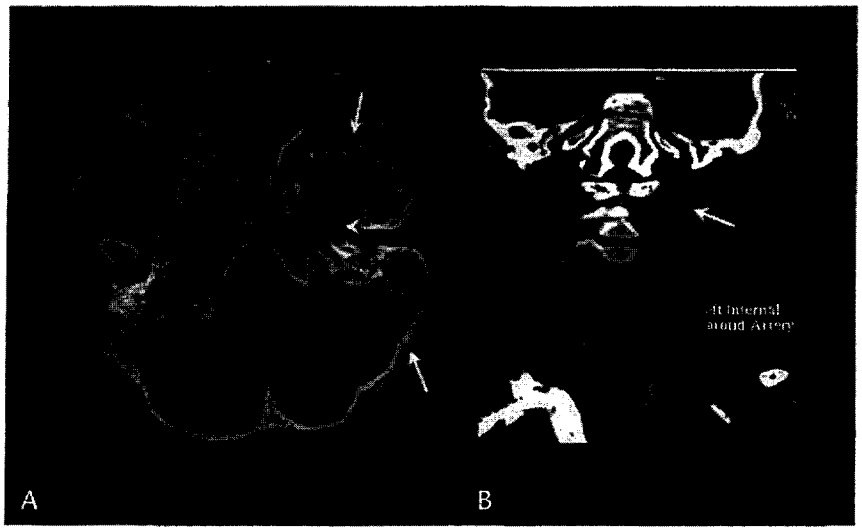


FIGURE 9. Images obtained from a 5-year-old boy. A, Computed tomographic image shows left-side skull base fractures involving left-side occiput, petrous bone, and sphenoid wing (arrows). Air densities are seen within the carotid canal (arrowhead). B, Computed tomography angiogram shows left-side cervical internal carotid arterial dissection with marked luminal narrowing (arrow).

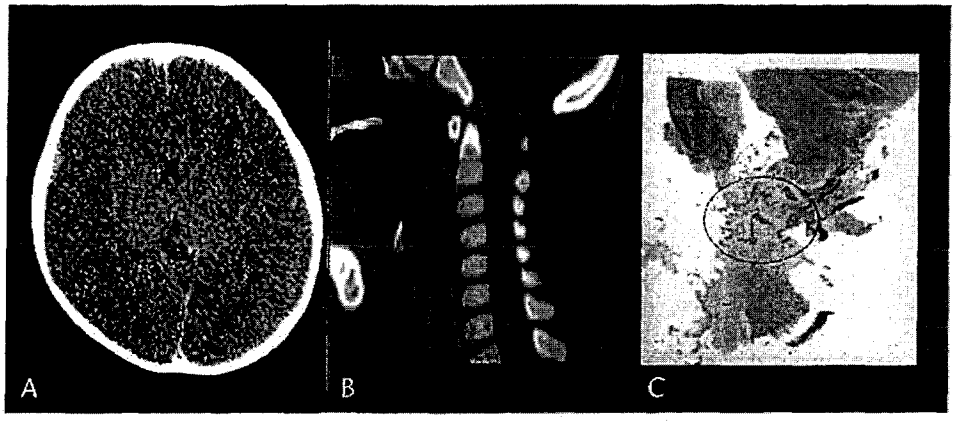


FIGURE 10. Images obtained from a 22-month-old boy with SCIWORA (caused by backward fall and parietal head impact) and hypoxic-ischemic injury and RHs. A, Axial brain CT image shows (1) bilateral cerebral low densities with decreased gray-white matter differentiation (edema) and (2) small high-density asymmetrical cerebral, extracerebral, and posterior interhemispheric hemorrhages. B, Sagittal reformatted cervical spinal computed tomographic image shows no spinal column abnormality (MRI not performed). C, Postmortem midsagittal section shows cervicomedullary disruption (circle). Diffuse hypoxic-ischemic axonal brain injury was also confirmed.

multiple lesions involving the cervical, thoracic, lumbar, or sacral level. The mechanisms of injury include hyperflexion, hyperextension, axial loading or rotation, and distraction. The range of spinal column and paraspinal injury includes vertebral or neural arch fractures, bony fragment or disk displacement, dislocations, instability, and paraspinal ligamentous, muscular, or vascular injury. Such injuries may not be apparent on plain films (eg, spinal cord injury without radiographic abnormality [SCIWORA]) and require additional CT plus MRI for complete evaluation.[129–131] Magnetic resonance imaging is particularly important for evaluating ligamentous injury and intraspinal injury. The range of intraspinal injury includes displaced bone or disk fragments and hematomas (eg, epidural) with spinal cord or nerve root compression. There may be edema, contusion, hemorrhage, transection of the spinal cord, or avulsion of 1 or more nerve roots. Computed tomographic angiography or MRA may be needed to evaluate vascular injury (eg, dissection). Cervical spinal cord injury may be associated with head injury or may be the unsuspected cause of respiratory failure and hypoxic-ischemic brain injury (eg, SCIWORA) (Fig. 10).[129–131] This should be evaluated by means of MRI in all such cases, whether AI or NAI. In addition, one must be aware of predisposing conditions that may result in major neurological deficits associated with *minor* head and neck trauma mechanisms (eg, craniocervical anomaly with instability Fig. 11; Chiari I malformation Fig. 12).

IMAGING ANALYSIS—COMPUTED TOMOGRAPHY

Regarding the initial computed tomographic examination, the findings are often nonspecific with regard to pattern of injury and timing and require a differential diagnosis (DDX). To properly analyze such a case from an imaging perspective, each injury component must be addressed separately, and then collectively, and then correlated with clinical and other data.[4,10,114] The major findings are often (1) extracerebral and cerebral high densities, (2) extracerebral isohypodensities, (3) cerebral low densities, with or without (4) scalp or skull abnormalities. In general, the DDX may include trauma (AI vs NAI), hypoxia-ischemia, ischemic injury (arterial vs venous occlusive disease), seizure edema, infectious or postinfectious conditions, coagulopathy, fluid-electrolyte derangement, metabolic or connective tissue disorder, and multifactorial.

Extracerebral high densities are often seen posteriorly along the tentorium, falx, interhemispheric fissure, and dural

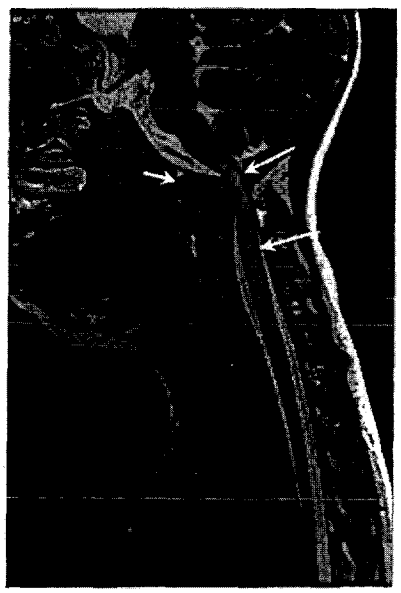


FIGURE 11. Image obtained from an 8-year-old girl with Down syndrome and minor trauma with quadriparesis. Sagittal T2 MRI scan shows hypoplastic dens, os odontoideum (anterior arrow), and anterior atlantoaxial instability (confirmed by means of CT) with cervicomedullary compression and high-intensity edema (posterior arrows).

FIGURE 12. Image obtained from a 3-year-old boy with Chiari I malformation, minor trauma, and subsequent quadriparesis. Sagittal T2 MRI scan shows cerebellar tonsils extending into the upper cervical canal (upper arrowhead) and diffuse high-intensity edema of the cervical spinal cord (lower arrows). No abnormality was present on plain films or CT (SCIWORA).

venous sinuses that may vary in laterality and symmetry (Figs. 2, 6, 7, 10, 13–16). These and other extracerebral high densities may be laminar, linear, nodular, or punctate. Using published criteria and timing parameters (discussed in the succeeding sections), these represent either acute to subacute hemorrhages (subarachnoid, subdural) or thromboses (eg, venous).[4,10,114–118] For apparent intracerebral high densities, it may be difficult to differentiate cerebral from SAHs (including those within the perivascular spaces) from vascular thromboses (eg, cortical, subependymal, or medullary venous thromboses). Computed tomography may not be able to distinguish focal or multifocal cerebral high densities as hemorrhagic contusion, hemorrhagic shear, or hemorrhagic infarction (Figs. 13, 16, 18). Extracerebral isohypodensities may represent subarachnoid spaces (eg, BECC), SDHG, hyperacute SDH, or chronic SDH (Figs. 14, 17). According to the literature, the timing for any of the mentioned findings is as follows: (1) hemorrhage or thromboses that are high density (ie, clotted) on CT (ie, acute to subacute) have a wide timing range of 3 hours to 7 to 10 days (Figs. 1, 2, 6, 7, 10, 13–18), (2) hemorrhage that is isohypodense on CT (ie, nonclotted) may be hyperacute (timing, <3 hours) or chronic (timing, >10 days) (Figs. 14, 17), (3) the low density may also represent preexisting wide, CSF-containing subarachnoid spaces (eg, BECC) or SDHG (ie, CSF containing) that may be acute or chronic (Figs. 14, 17), (4) blood levels are unusual in the subacute unless there is coagulopathy (Fig. 7), (5) CT cannot distinguish acute hemorrhage from rehemorrhage on existing chronic collections (BECC or chronic SDHG) (Fig. 17), and (6) the interhemispheric SDH is no longer considered characteristic of NAI (Figs. 2, 6, 7, 13–16).[4,10,114–118]

Cerebral low densities may vary in bilaterality and symmetry and be associated with decreased gray-white matter differentiation or mass effect (Figs. 2, 10, 17). In general, this indicates edema/swelling, the timing of which depends on causation. If related to trauma, such edema/swelling may represent primary injury or secondary injury and be acute-hyperacute (eg, timing of few hours) or delayed (eg, timing of several hours to a few days), including association with lucid interval and short falls.[4,10,114,123–126] Bilateral diffuse edema is most commonly observed in hypoxia-ischemia but may also be observed in other diffuse processes (eg, fluid-electrolyte imbalance, status epilepticus, encephalitis, etc). Focal or multifocal edema may be observed in contusion (eg, gray matter), shear (eg, white matter), infarction (gray or white matter), encephalitis, or demyelination (eg, acute disseminated encephalomyelitis).

Cranial defects may represent Fx, and their timing range is very broad (eg, hours to months old) (Fig. 1).[4,10,114] Furthermore, Fx morphology (eg, multiple, growing) does not reliably distinguish accidental from nonaccidental causation. Scalp collections (hemorrhage, edema, blood level) are also nonspecific with regard to causation and timing (Fig. 1).[4,10,114] If caused by trauma, the timing range is also rather broad (eg, hours to days old). Sutural widening may indicate diastatic Fx

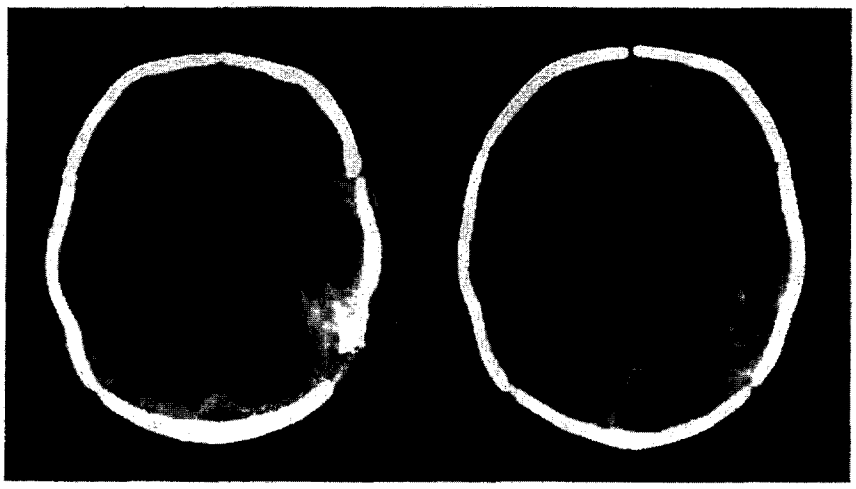


FIGURE 13. Images obtained from a 1-day-old female infant delivered by means of spontaneous vaginal delivery and with subsequent apneic episodes. Computed tomography demonstrates left-side temporal cerebral and extracerebral high-density hemorrhage (or thromboses); high-density hemorrhage is also demonstrated along the interhemispheric fissure, tentorium, and dural venous sinuses. The results of coagulopathy test and sepsis workup were negative (final diagnosis, birth trauma?).

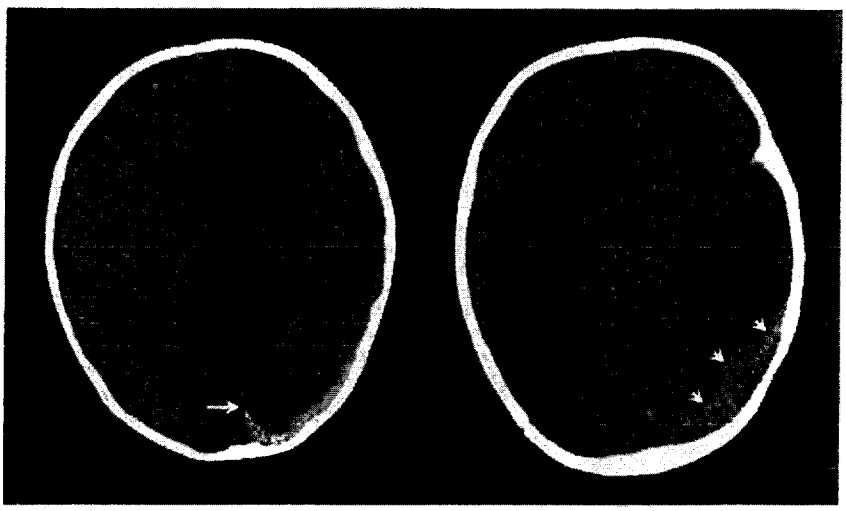


FIGURE 14. Images obtained from a 4-month-old male infant with 2-week viral illness who progressed to septic shock (*Staphylococcus aureus*), endocarditis, severe mitral regurgitation, and coagulopathy. Noncontrast axial CT images show high-density extracerebral hemorrhages (and/or thromboses) along the left-side tentorium, dural venous sinuses, falx, and interhemispheric fissure (arrows). In this case, the bifrontal low-density extracerebral spaces likely represent slightly prominent infantile subarachnoid spaces (BECC?) or underdevelopment, rather than chronic SDH or subdural hygroma.

or increased intracranial pressure. Accessory sutures or synchondroses and developmental fissures may mimic Fx. Intrasutural bones (eg, wormian) may be associated with a skeletal dysplasia or metabolic disorder (Fig. 4).

Subsequent or follow-up computed tomographic examinations may show surgical changes (eg, postevacuation, ventricular catheter, pressure-monitoring device), evolving, redistributing, or recurrent/new hemorrhages, and evolving cerebral densities (edema/swelling). Subsequent CT examinations during the weeks or months may show evolution to permanent cerebral tissue loss (ie, atrophy, encephalomalacia).

IMAGE ANALYSIS—MAGNETIC RESONANCE IMAGING

On an imaging basis, only MRI may provide more precise information regarding pattern of injury and timing, particularly with regard to (1) hemorrhage versus thromboses, and (2) brain injury. The MRI should be performed as soon as feasible, and the findings be compared with the findings from the earlier CT. As a result, MRI has become the standard for such evaluation in these matters.[4,10,114–117,121,123–126]

Hemorrhages and Thromboses

Using published MRI guidelines (Table 1), in general, the evolutionary timing for hemorrhages or thromboses (eg, venous) are as follows: (1) hyperacute phase (timing, <12 hours): T1 isohypointense, T2 hyperintense; (2) acute phase (timing, 1–3 days): T1 isohypointense, T2 hypointense; (3) early subacute phase (timing, 3–7 days): T1 hyperintense, T2 hypointense; (4) late subacute phase (timing, 7–14 days): T1 hyperintense, T2 hyperintense; (5) early chronic phase (timing, >14 days): T1 hyperintense, T2 hyperintense; (6) late chronic phase (timing, >1 to 3 months): T1 isohypointense, T2 hypointense.[4,10,114–117,121,123–124] Mixed intensity collections are problematic regarding timing. Matching the MRI findings with the computed tomographic findings may help, along with follow-up MRI. Blood levels may indicate subacute hemorrhage versus coagulopathy. The timing guidelines are better applied to the sediment than to the supernatant. In addition, a single MRI may not reliably differentiate T1-hypointense/T2-hyperintense collections as representing CSF collections (eg, BECC, acute SDHG) versus hyperacute SDH versus chronic collections (SDH, SDHG). Gradient-recalled echo hypointensities are iron sensitive but do not assist with timing unless matched with

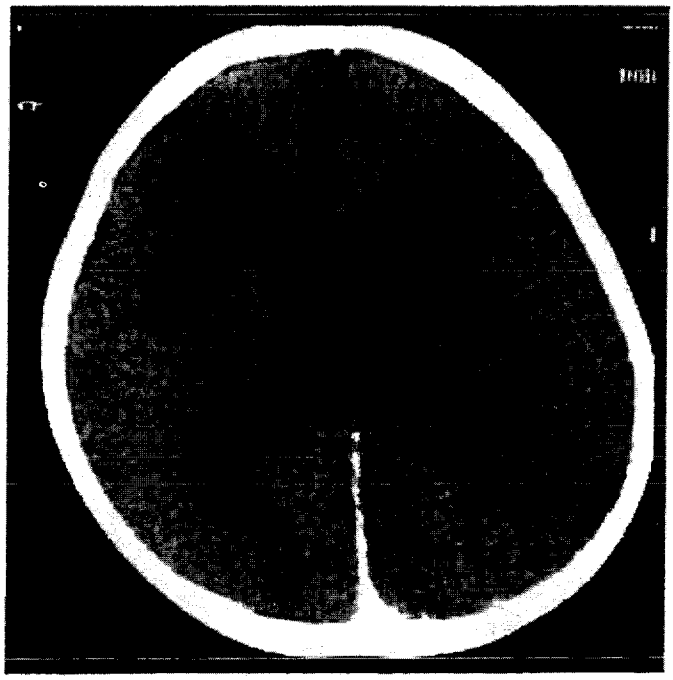


FIGURE 15. Image obtained from a 23-month-old girl who had recent viral gastrointestinal illness, ALTE, RHs, then brain death. Computed tomographic image shows posterior interhemispheric high densities at the level of portions of the inferior sagittal, straight, and superior sagittal sinuses, plus poor cerebral gray-white matter differentiation and moderate ventriculomegaly. Autopsy showed extensive dural and cerebral venous sinus thrombosis with extensive hypoxic-ischemic diffuse axonal brain injury.

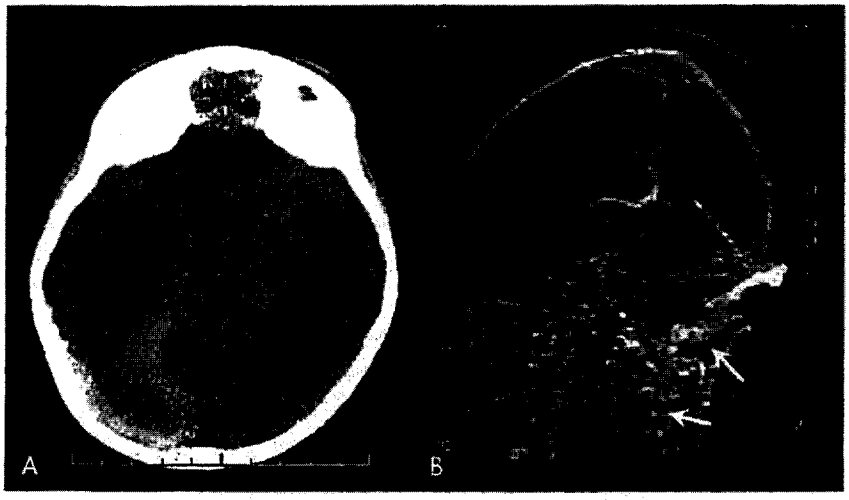


FIGURE 16. Images obtained from a 19-month-old boy who had 1 week of febrile illness (treated with antibiotics), followed by ALTE with RHs. A, Computed tomographic image shows high-density hemorrhages (or thromboses) along the right tentorium and dural venous sinuses. B, Magnetic resonance imaging with MRV shows irregular flow gaps with incomplete opacification of the right-side internal jugular vein and sigmoid sinus. Other flow gaps were demonstrated within the superior sagittal and straight sinuses, along with multiple venous collaterals (diagnosis, DVST).

T1, T2, and computed tomographic densities. Gradient-recalled echo and other magnetic susceptibility sequences are also sensitive to venous thromboses (eg, cortical, medullary, subependymal) that are not detected by means of MRV.

Brain Injury

With regard to brain injury, MRI may distinguish hypoxic-ischemic injury (diffuse relatively symmetrical DWI/ADC restricted diffusion with or without matching T1/T2 abnormalities) from shear and contusional injury (focal/multifocal restricted diffusion, GRE hypointensities, with T2/FLAIR edema). Shear and contusional injury, however, may not be reliably differentiated from focal/multifocal ischemic or hemorrhagic infarction (eg, dissection, vasculitis, venous, embolic) without supportive MRA, CTA, MRV, or angiography.[4,10,114,123–125] In addition, similar cortical or subcortical intensity abnormalities (including restricted diffusion) may also be observed in encephalitis, seizures, and metabolic disorders. Using published MRI criteria and parameters,[114,123–126] in general, the evolutionary timing for ischemic injury is as follows: (1) hyperacute phase (timing, <1 day): DWI hyperintense, ADC hypointense; MRS result, lactate peak; (2) early acute phase (timing, 1–2 days): additional T2 hyperintensity; (3) late acute phase (timing, 2–4 days): additional T1 hyperintensity; (4) early subacute phase (timing, 6–7 days): additional T2 hypointensity; (5) late subacute phase (timing, 7–14 days): additional DWI isohypointense, ADC isohyperintense; (6) chronic phase (timing, >14 to 21 days): additional atrophy. If related to trauma, focal/multifocal ischemic findings may be caused by arterial injury (eg, dissection), venous injury (eg, tear, thrombosis), arterial spasm (as with any cause of hemorrhage), herniation, or edema with secondary perfusion deficit or seizures (eg, status epilepticus). Hypoxia-ischemic brain injury caused by apnea/respiratory arrest may occur with head trauma or with neck/cervical spine/cord injuries (eg, SCIWORA), whether AI or NAI.[114,123,129–131] It may also occur with any nontraumatic cause (eg, choking, paroxysmal coughing, aspiration).[132] In addition to the diffuse brain injury, there may be associated subarachnoid and subdural hemorrhage without mass effect.[104–109]

CONDITIONS MIMICKING NONACCIDENTAL INJURY

Traumatic and nontraumatic conditions may mimic the clinical presentations (ie, the triad) and imaging findings of NAI. These include accidental trauma (as previously discussed), birth trauma, hypoxia-ischemia, cardiopulmonary

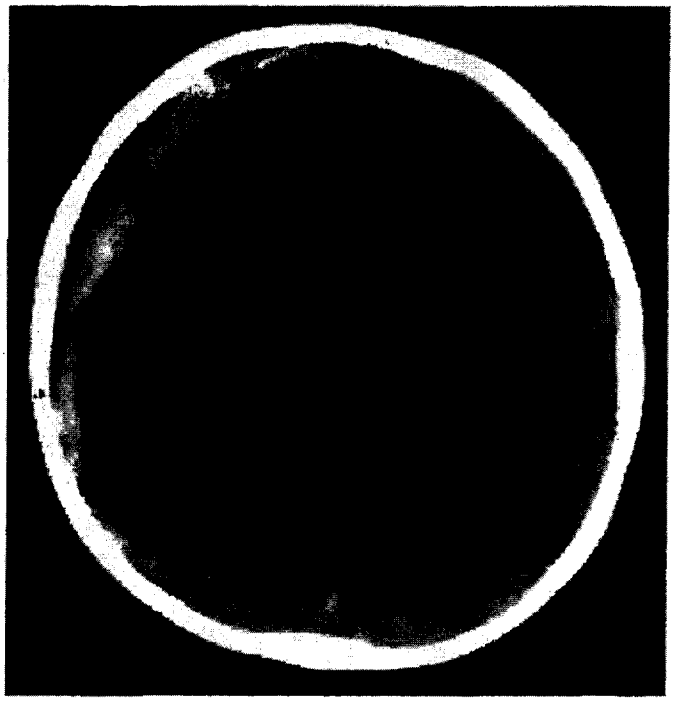


FIGURE 17. Image obtained from an 8-month-old male infant who had ALTE, right-side occipital skull fracture (not shown), a healing right-side distal radial fracture, and then had brain death. Computed tomographic image shows a right-side, mixed density extracerebral collection with right-side cerebral low density, mass effect, and leftward shift. High-density hemorrhages (or thromboses) are also present along the tentorium. There was disagreement among the forensic experts regarding hyperacute-acute SDH versus chronic SDH with rehemorrhage.

resuscitation, infectious or postinfectious conditions (eg, sepsis, meningoencephalitis, postvaccinial), vascular diseases, coagulopathies, venous thrombosis, metabolic disorders, neoplastic processes, certain therapies, extracorporeal membrane oxygenation (ECMO), and other conditions.[4,5,10,114,115,133] Regarding the pathogenesis of the triad (with and without other organ system involvement [eg, skeletal]), and whether caused by NAI, AI, or nontraumatic etiologies, the pathophysiology seems to be some combination or sequence of factors, including increased intracranial pressure, increased venous pressure, systemic hypotension or hypertension, vascular fragility, hematologic derangement, and/or collagenopathy superimposed on the immature CNS and other systems.[107,115,123,132–146]

Although the initial medical evaluation, including history, laboratory tests, and imaging studies, may suggest an alternative condition, the diagnosis may not be made because of a *rush to judgment* regarding NAI. It is important to be aware of these mimics because a more extensive workup may be needed beyond the routine *screening* tests. In addition, the lack of confirmation of a specific condition does not automatically indicate the *default* diagnosis of NAI. In all cases, it is critical to review all records dating back to the pregnancy and birth, the postnatal pediatric records, the family history, the more recent history preceding the short-term presentation, the details of the short-term event itself, the resuscitation, and the subsequent management, all of which may contribute to the clinical and imaging findings.[4,5,10,115,133]

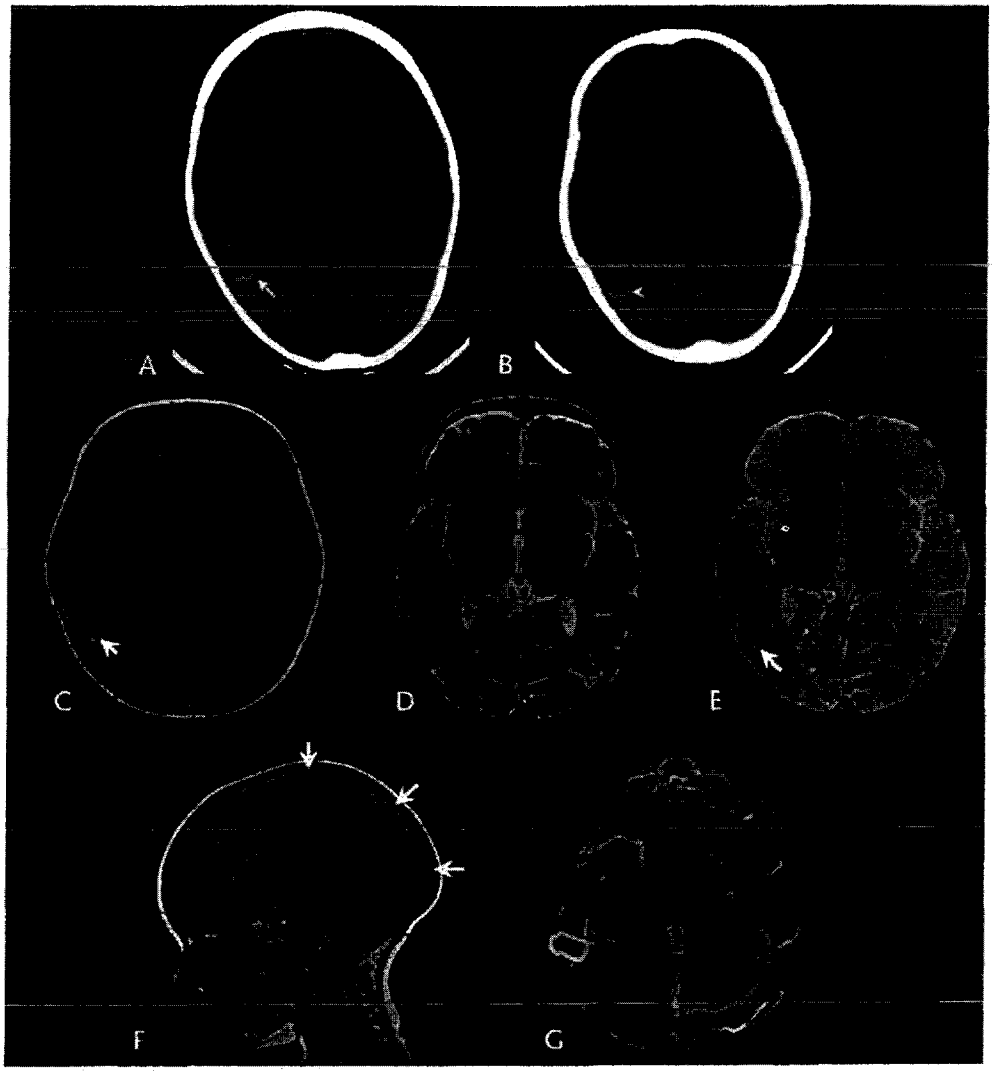


FIGURE 18. Images obtained from a 22-month-old boy who experienced lethargy, vomiting, and seizures after a viral illness, plus thrombocytopenia and iron deficiency anemia. A–B, Computed tomographic images show right-side posterior temporal and peritentorial high-density foci of hemorrhage or thrombosis (arrows). Axial T1 (C), T2 (D), and GRE (E) images show corresponding T1-hyperintense and GRE-hypointense foci with associated T2 hyperintensity (arrows). F, Sagittal T1 MRI scan shows hyperintensity along the superior sagittal sinus (arrows [thrombosis vs slow flow]). G, Axial MRV projection image shows nonvisualization of the superior sagittal, right-side transverse, and right-side sigmoid sinuses (diagnosis, postviral dural and cerebral venous thrombosis [extensive coagulopathy workup continues]).

A recent review presented by Sirotnak[133] extensively catalogues the many conditions that may mimic abusive head trauma. These include perinatal conditions (birth trauma and congenital malformations), accidental trauma, genetic and metabolic disorders, hematologic diseases and coagulopathies, infectious diseases, autoimmune and vasculitic conditions, oncological disease, toxins, poisons, nutritional deficiencies, and medical and surgical complications. The reader is encouraged to read this review.[133] An abbreviated discussion is presented in this article along with some examples.

Birth Trauma and Neonatal Conditions

Manifestations of birth trauma, including Fx, SDH, and RH, may persist beyond the neonatal period and mimic CNS findings of abuse.[145–151] Other examples are the cases of infants following ECMO therapy, at-risk preterm neonates, and infants with congenital heart disease.[4,5,10,123,124,152] When evaluating the condition of a young infant with apparent NAI, it is important to consider that the clinical and imaging findings may actually stem from parturitional and neonatal issues. This includes hemorrhage or rehemorrhage into collections existing at birth (Figs. 5, 8, 13).

Developmental Anomalies

Vascular malformations of the CNS in neonates and infants are relatively rare.[115,133,153,154] The most common are the vein of Galen malformations. Aneurysms are also rare in childhood but may arise within the circle of Willis. Aneurysms outside the circle are usually mycotic or traumatic in origin. Increased risk of aneurysm is associated with certain conditions, such as coarctation of the aorta, polycystic kidney disease, neurofibromatosis, and a family history positive for aneurysm. A number of syndromes in childhood are associated with vascular anomalies and may present with intracranial hemorrhage. These syndromes include, as examples, PHACE (*p*osterior fossa brain malformations, *h*emangiomas, *a*rterial anomalies, *c*oarctation of the aorta, *c*ardiac defects, and *e*ye abnormalities), Sturge-Weber, Beckwith-Wiedemann, Klippel-Trenaunay-Weber, Maffucci, and Olser-Weber-Rendu. Arachnoid cysts are also known to be associated with SDH and RH, spontaneously and with trauma (Fig. 19).[133,155]

Genetic and Metabolic Disorders

A number of conditions in this category may present with intracranial hemorrhage (eg, SDH) or RH. These include osteogenesis imperfecta, glutaric aciduria type I, Menkes kinky hair disease, Ehlers-Danlos and Marfan syndromes, homocystinuria, and others (Fig. 19).[115,133,135,136,156]

Hematologic Disease and Coagulopathy

Many conditions in this category predispose to intracranial hemorrhage and RH.[4,5,10,114,115,133,140–143,157] The bleeding or clotting disorder may be primary or

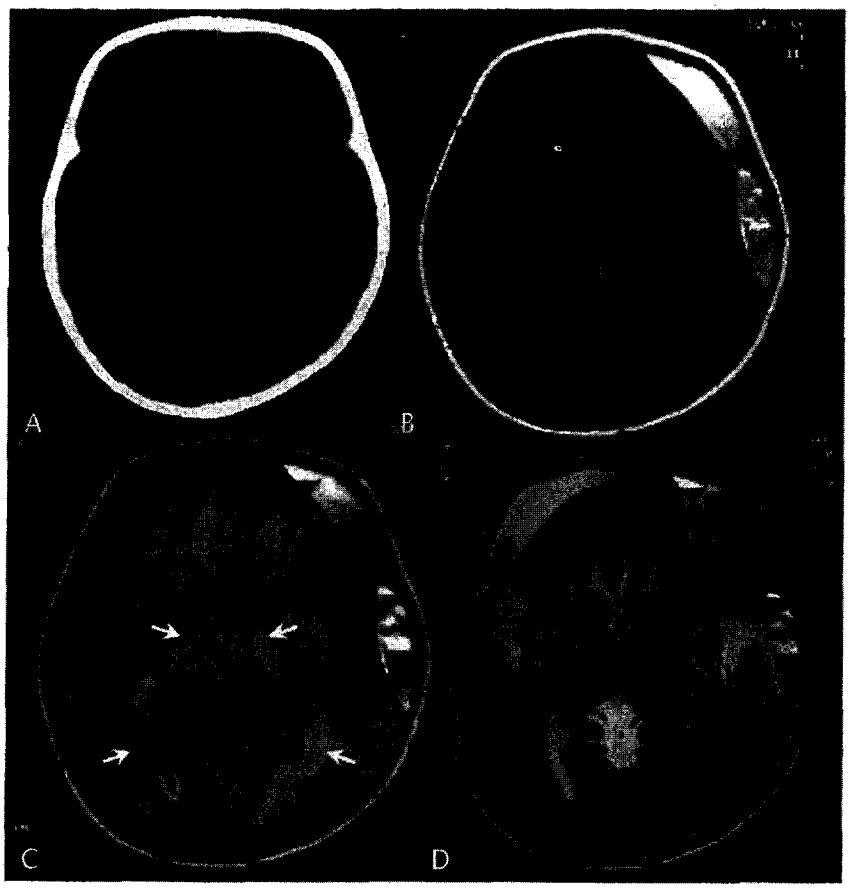


FIGURE 19. Images obtained from a 9-month-old male infant with glutaric aciduria type 1, SDHs, and RHs. CT (A), T1 (B), FLAIR (C), and T2 (D) MRI images show bilateral mixed-density and mixed-intensity extracerebral collections with fluid levels and septations, especially on the left side. Other characteristic findings for glutaric aciduria type 1 include bilaterally wide sylvian fissures (arachnoid cysts) plus abnormal basal ganglia (globus pallidus) and cerebral white matter intensities (arrows).

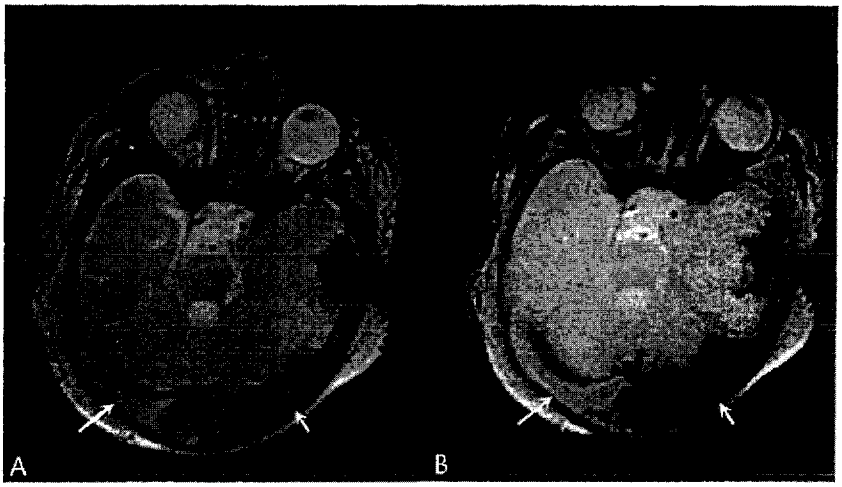


FIGURE 20. Images obtained from a 1-week-old male neonate with seizures, thrombocytopenia, antithrombin III deficiency, and ECMO for pulmonary hypertension. Axial T2 FSE (A) and GRE (B) MRI images show bilateral, mixed-intensity SDHs (arrows).

secondary (Figs. 7, 14–16, 18, 20, 21). In some cases, a more extensive workup beyond the usual *screening* tests will be needed, including a hematology consultation. Included in this category are the anemias, hemoglobinopathies (eg, sickle cell disease), hemorrhagic disease of the newborn (vitamin K deficiency Fig. 21), hemophilia A and B, factor V and XII deficiencies, von Willebrand disease, idiopathic thrombocytopenic purpura, disseminated intravascular coagulation and consumption coagulopathy associated with other conditions (eg, trauma, infection), liver disease, nephrotic syndrome, hemophagocytic lymphohistiocytosis, anticoagulant therapy, and others. Venous thrombosis may involve the dural venous sinuses (ie, dural venous sinus thrombosis [DVST]) and/or the cerebral veins (ie, cerebral vein thrombosis [CVT]) and be associated with primary or secondary hematologic or coagulopathic state.[10,123,124,133,158–161] Risk factors include acute systemic illness, dehydration (fluid-electrolyte imbalance), sepsis, perinatal complications, chronic systemic disease, cardiac disease, connective tissue disorder, hematologic disorder, oncological disease and therapy, head and neck infection, and hypercoagulable states. Seizure and/or neurological deficit are common, and hemorrhagic infarction is characteristic. Subarachnoid hemorrhage, SDH, or RH may also be observed, especially in infants (Figs. 15, 16, 18, 22). Relative high densities anywhere along the dural venous sinuses, tentorium, and falx (interhemispheric fissure and inferior sagittal sinus) may be seen on initial CT. Linear high densities may also be present along the distribution of the cortical ("cord sign"), subependymal, or medullary veins and give the impression of SAH, SDH, or intracerebral

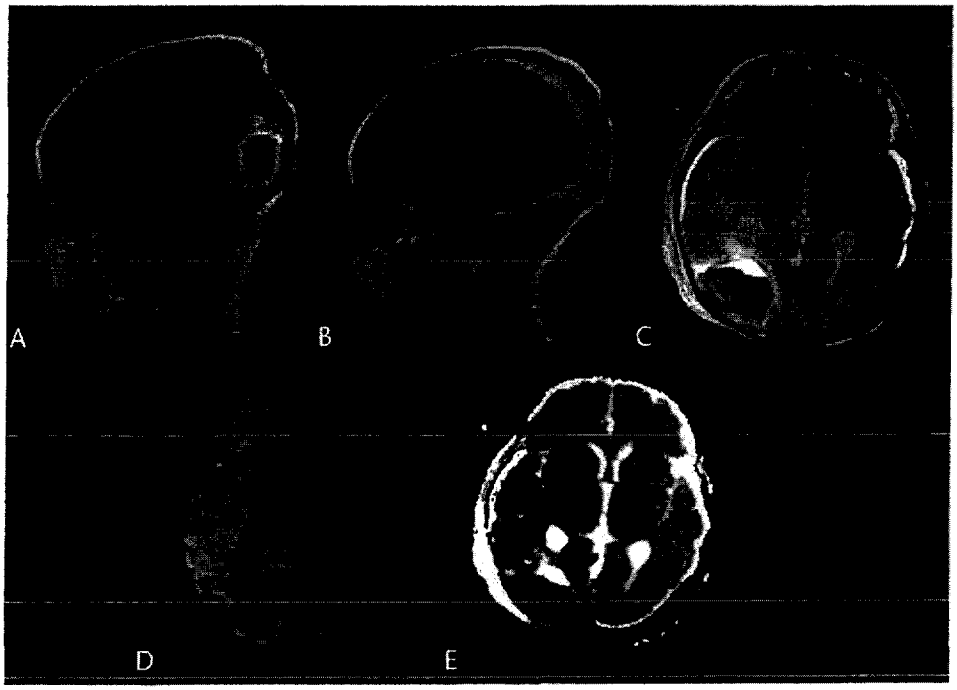


FIGURE 21. Images obtained from a 1-week-old male neonate who had seizures after delivery at home (no vitamin K administered). After surgical evacuation of large, right-side SDH, sagittal T1 (A, B), axial T2 (C), ADC (D), and DWI (E) images show bilateral mixed-intensity extracerebral and intracerebral hemorrhages and right-side cerebral hemispheric restricted diffusion (likely infarction) (diagnosis, hemorrhagic disease of the newborn [vitamin K deficiency]).

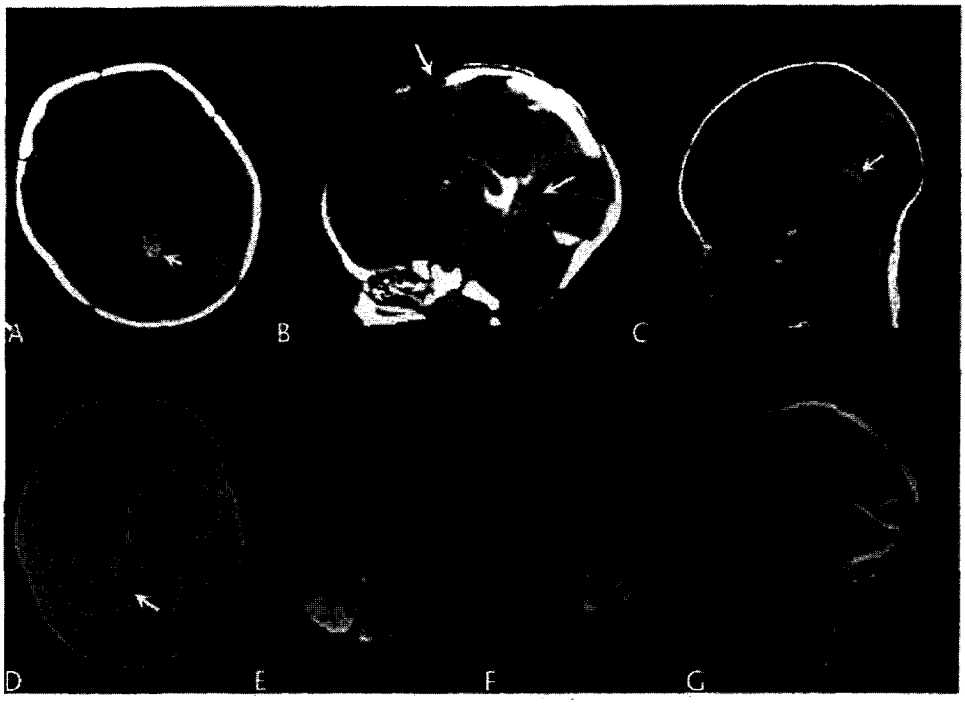


FIGURE 22. Images obtained from a 2-week-old male neonate with lethargy in emergency room (ER). Computed tomographic image (A) shows a focal midline hyperdensity at the level of the straight sinus (arrowhead). Sagittal CTV image (B) shows luminal masses along the straight and superior sagittal sinuses (arrows). Sagittal T1 (C) and axial GRE (D) images show the thrombus within the straight sinus (arrows). Axial DWI images (E–F) show restricted diffusion in multiple cortical areas (likely infarction vs suppuration). Magnetic resonance venography (G) is of poor diagnostic quality as compared with CTV (diagnosis, group B streptococcal meningitis with DVST).

hemorrhage. The "empty delta" sign may be seen within the superior sagittal sinus on contrast-enhanced CT. There may be multifocal infarctions (hemorrhagic or nonhemorrhagic) or intraventricular hemorrhage. With extensive dural venous sinus or cerebral venous thrombosis, there may be massive, focal, or diffuse edema. Orbit, paranasal sinus, or otomastoid disease may be associated with basal venous sinus thrombosis (eg, cavernous, petrosal, sphenoparietal). The thromboses and associated hemorrhages have variable MRI appearance depending on their age (see Image Analysis–Magnetic Resonance Imaging section and Table 1). Computed tomographic venography or MRV may readily detect DVST but not cerebral vein thrombosis, which may be suspected because of the characteristic distribution of hemorrhage or thromboses along venous structures, as demonstrated on susceptibility-weighted sequences (eg, GRE hypointensity). Depending on the clinical context, treatment may be directed only to the specific cause (eg, infection) or may also include anticoagulation or thrombolysis.

Infectious and Postinfectious Conditions

Meningitis, encephalitis, or sepsis (eg, bacterial, viral, granulomatous, parasitic) may involve vascular structures resulting in vasculitis, arterial or venous thrombosis, mycotic aneurysm, infarction, and hemorrhage (Figs. 3, 14–17, 22, 23). Subdural hemorrhage and RH may also be observed.

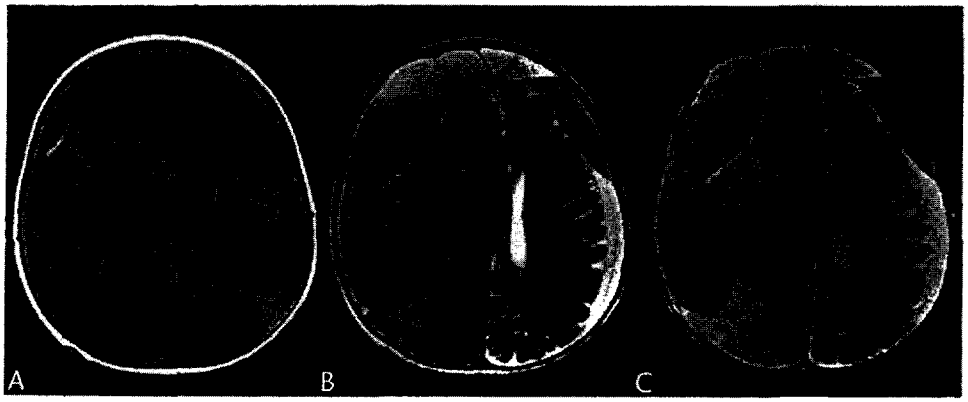


FIGURE 23. Images obtained from a 5-month-old male infant who had macrocephaly and seizures after having group D streptococcal (nonenterococcal) meningitis at the age of 3 days. Axial T1 (A), T2 (B), and GRE (C) images show bilaterally large and mixed-intensity extracerebral collections with septations and asymmetrical mass effect (likely chronic subdural effusions or hygromas with rehemorrhage).

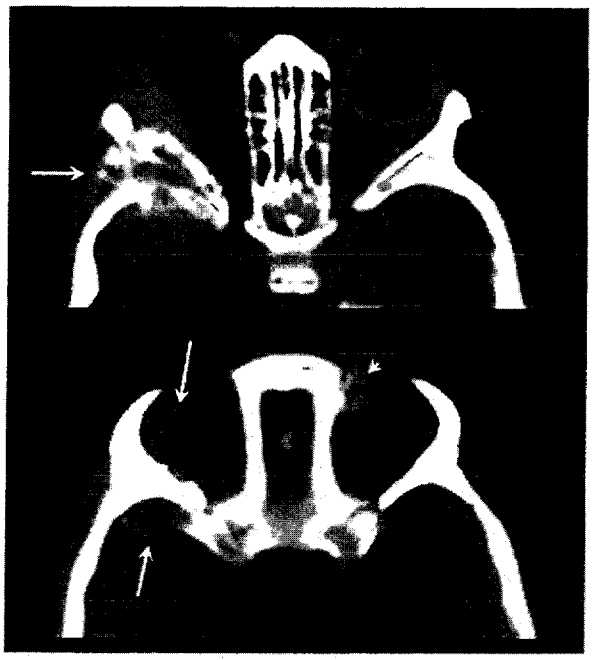


FIGURE 24. Images obtained from an 18-month-old girl with periorbital and facial ecchymoses in ER, evaluated for NAI. Computed tomographic image shows bilateral iso-high-density orbital soft tissue masses with bone destruction (arrows) and extension into the right-side middle cranial fossa (diagnosis, neuroblastoma).

Postinfectious illnesses (eg, postvaccinial) may also be associated with these findings.[139] Included in this category are the *encephalopathies of infancy and childhood* and *hemorrhagic shock and encephalopathy syndrome*.[115,133]

Autoimmune and Vasculitic Conditions

These include Kawasaki disease, systemic lupus erythematosis, moyamoya disease, Wegener granulomatosis, and Behçet syndrome.[115,133]

Oncological Disease

Hematologic malignancies, solid tumors of childhood, and their attendant therapies (including transplantation) are commonly associated with a variety of sequelae or complications that predispose to hemorrhage (eg, SDH and RH).[115,133] This includes vascular invasion by tumor, immunocompromise, infection, and coagulopathy. The clinical presentation and image findings may be mistaken for NAI (eg, leukemia, neuroblastoma) (Fig. 24).

Toxins, Poisons, and Nutritional Deficiencies

This category includes lead poisoning, cocaine, anticoagulants, and vitamin deficiencies (eg, vitamins K, C, D) (Figs. 21, 25). Preterm neonates and other chronically ill infants are particularly vulnerable to nutritional deficiencies and complications of prolonged immobilization that often primarily affect bone development. Such infants may have skeletal imaging findings (eg, multiple healing fractures) that are misinterpreted as NAI, particularly if they present with AI that is complicated by SDH and RH (Fig. 25).[162–174]

Medical and Surgical Complications

This category includes (1) anticoagulant therapy or treatment-induced coagulopathy and (2) morbidity from medical or surgical interventions.[115,133]

CONCLUSIONS

In view of the currently available data, it is clear that we do not have an established EBM platform from which to

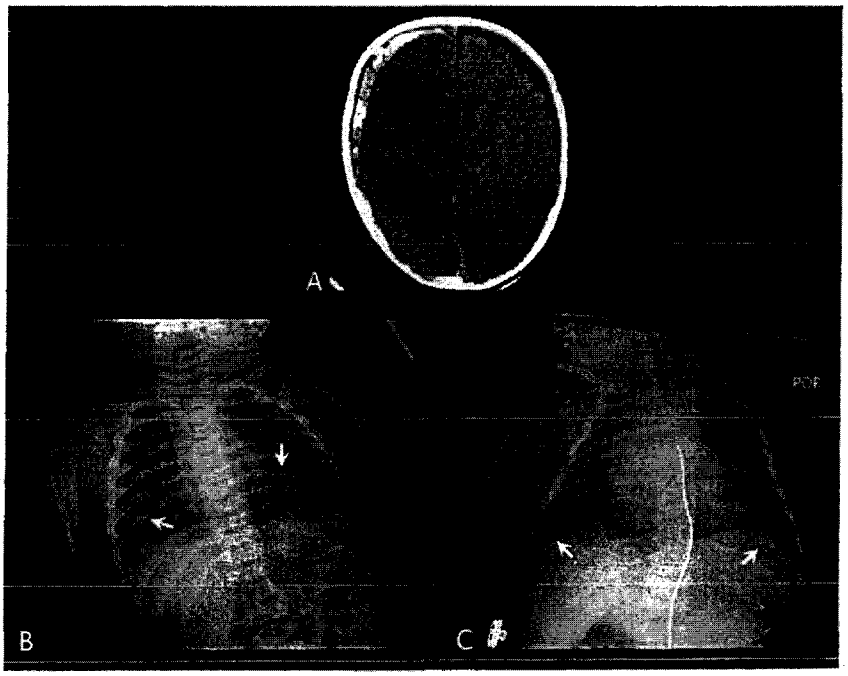


FIGURE 25. Images obtained from a 7-month-old male infant (25-week preterm birth) dropped with head impact to floor, RHs, evaluated in ER. Computed tomographic image (A) shows right-side mixed high-density extracerebral collection, left-side low-density extracerebral collection, posterior interhemispheric high-density hemorrhage, and right-side cerebral low-density edema. Chest radiograph in ER (B) shows bilateral anterior and posterior old, healing rib fractures. Comparison with earlier chest radiograph (C) at discharge from neonatal intensive care unit shows diffuse osteopenia and anterior rib flaring (arrows). Diagnosis: rickets of prematurity vs NAI?; AI with acute SDH superimposed on BECC vs NAI?

distinguish NAI from AI and, in some cases, traumatic from nontraumatic CNS injury. More reliable research is needed to establish a sound scientific foundation for CNS injury in NAI. The young infant is assumed more vulnerable to traumatic CNS injury, whether accidental or not, as compared with the older child or adult, and relies on the attention of caretakers for safety. However, as the infant becomes more mobile (rolling, crawling, walking, etc), the risk of AI (eg, from falls) increases. Furthermore, the interaction with older siblings or other children becomes a factor. The medical and imaging findings cannot diagnose intentional injury. Only the child protection investigation may provide the basis for inflicted injury in the context of supportive medical, imaging, or pathological findings. Furthermore, biomechanical factors must be taken into consideration regarding the mechanism of trauma.

The radiologist should describe the imaging findings in detail, including the pattern, distribution, and severity of injury. A DDX is given, and timing ranges are provided if possible. If NAI is at issue, then the radiologist must directly communicate the imaging findings to the primary care team and be available to consult with child protection services and other medical or surgical consultants, including the pathologist or biomechanical specialist, law enforcement investigators, and attorneys for all parties, as appropriate.[1–5] The pattern of injury and the timing parameters, as may be provided by MRI, are particularly important with regard to correlation of events as reported by witnesses and potential suspects. The radiologist must also be aware of certain conditions that are known to have clinical and imaging features that may mimic abuse.[1–5] These should be properly ruled out, and the possibility of combined or multifactorial mechanisms with synergistic effects should also be considered (eg, predisposing condition plus trauma). A timely and thorough multidisciplinary evaluation may be the difference between an appropriate child protection and an improper breakup of the family or a wrongful indictment and conviction.

REFERENCES

1. Kraus J, Fife D, Cox P, et al. Incidence, severity, and external cause of pediatric brain injury. *Am J Dis Child.* 1986;140:687–693.
2. Zimmerman RA, Bilaniuk L. Pediatric head trauma. *Neuroimaging Clin N Am.* 1994;4:349–366.
3. Bruce DA, Zimmerman RA. Shaken impact syndrome. *Pediatr Ann.* 1989;18:482–494.
4. Kleinman P, Barnes P. Head trauma. In: Kleinman P, ed. *Diagnostic Imaging of Child Abuse.* 2nd ed. New York, NY: Mosby Year Book; 1998:285–342.
5. Frasier L, Rauth Farley K, Alexander R, et al. *Abusive Head Trauma in Infants and Children: A Medical, Legal, and Forensic Reference.* St Louis, MO: GW Medical Publishing; 2006.
6. Harding B, Risdon RA, Krous HF. Shaken baby syndrome [editorial]. *BMJ.* 2004;328:720–721. Cited: American Academy of Pediatrics Committee on Child Abuse and Neglect. Shaken baby syndrome: inflicted cerebral trauma. *Pediatrics.* 1993;92:872–875.
7. Kempe CH, Silverman FN, Steele BF, et al. The battered child syndrome. *JAMA.* 1962;181:17–24.
8. Caffey J. On the theory and practice of shaking infants. Its potential residual effects of permanent brain damage and mental retardation. *Am J Dis Child.* 1972;124:161–169.
9. Silverman FN. Unrecognized trauma in infants, the battered child syndrome, and the syndrome of Ambroise Tardieu. Rigler lecture. *Radiology.* 1972;104:337–353.
10. Barnes P. Ethical issues in imaging nonaccidental injury: child abuse. *Top Magn Reson Imaging.* 2002;13:85–94.
11. Hymel KP, Bandak FA, Partington MD, et al. Abusive head trauma? A biomechanics-based approach. *Child Maltreat.* 1998;3:116–128.
12. American Academy of Pediatrics: Committee on Child Abuse and Neglect. Shaken baby syndrome: rotational cranial injuries—technical report. *Pediatrics.* 2001;108:206–210.
13. Case ME, Graham MA, Handy TC, et al. Position paper on fatal abusive head injuries in infants and young children. *Am J Forensic Med Pathol.* 2001;22:112–122.
14. Donohoe M. Evidence-based medicine and shaken baby syndrome, part I: literature review, 1966–1998. *Am J Forensic Med Pathol.* 2003;24:239–242.
15. Feldman KW, Bethel R, Shurgerman RP, et al. The cause of infant and toddler subdural hemorrhage: a prospective study. *Pediatrics.* 2001;108;636–646.
16. Duhaime AC, Alario AJ, Lewander WJ, et al. Head injury in very young children: mechanisms, injury types, and ophthalmologic findings in 100 hospitalized patients younger than 2 years of age. *Pediatrics.* 1992;90:179–185.
17. Zimmerman RA, Bilaniuk LT, Bruce D, et al. Interhemispheric acute subdural hematoma. A computed tomographic manifestation of child abuse by shaking. *Neuroradiology.* 1979;16:39–40.
18. Merten DF, Osborne DRS, Radkowski MA, et al. Craniocerebral trauma in the child abuse syndrome: radiological observations. *Pediatr Radiol.* 1984;14:272–277.
19. Greenberg J, Dohen WA, Cooper PR. The hyperacute extra-axial intracranial hematoma: computed tomographic findings and clinical significance. *Neurosurg.* 1985;17:48–56.
20. Cohen RA, Kaufman RA, Myers PA, et al. Cranial computed tomography in the abused child with head injury. *AJNR Am J Neuroradiol.* 1985;6:883–888.
21. Alexander RC, Schor DP, Smith WL Jr. Magnetic resonance imaging of intracranial injuries from child abuse. *J Pediatr.* 1986;109:975–979.
22. Bird CR, McMahan JR, Gilles RH, et al. Strangulation in child abuse: CT diagnosis. *Radiology.* 1987;163:373–375.
23. Sato Y, Yuh WT, Smith WL, et al. Head injury in child abuse: evaluation with MR imaging. *Radiology.* 1989;173:653–657.
24. Ball WS Jr. Nonaccidental craniocerebral trauma (child abuse): MR imaging. *Radiology.* 1989;173:609–610.
25. Hart BL, Dudley MH, Zumwalt RE. Postmortem cranial MRI and autopsy correlation in suspected child abuse. *Am J Forensic Med Pathol.* 1996;17:217–224.
26. Hymal KP, Rumack CM, Hay TC, et al. Comparison of intracranial CT findings in pediatric abusive and accidental head trauma. *Pediatr Radiol.* 1997;27:743–747.
27. Haseler LJ, Arcnue E, Danielsen ER, et al. Evidence from proton MR spectroscopy for a metabolic cascade of neuronal damage in shaken baby syndrome. *Pediatrics.* 1997;99:4–14.
28. Feldman KW, Weinberger E, Milstein JM, et al. Cervical spine MRI in abused infants. *Child Abuse Negl.* 1997;21:199–205.
29. Mogbo KI, Slovis TL, Canady AI, et al. Appropriate imaging in children with skull fractures and suspicion of abuse. *Radiology.* 1998;208:521–524.
30. Petitti N, Williams DW. CT and MRI of nonaccidental pediatric head trauma. *Acad Radiol.* 1998;5:215–223.
31. Dias MS, Backstrom J, Falk M, et al. Serial radiography in the infant shaken impact syndrome. *Pediatr Neurosurg.* 1998;29:77–85.
32. Ewing-Cobbs L, Kramer L, Prasad M, et al. Neuroimaging, physical, and developmental findings after inflicted and noninflicted traumatic brain injury in young children. *Pediatrics.* 1998;102:300–307.
33. Rooks VJ, Sisler C, Burton B. Cervical spine injury in child abuse: report of two cases. *Pediatr Radiol.* 1998;28:193–195.
34. Rao P, Carty H, Pierce A. The acute reversal sign: comparison of medical and nonaccidental injury patients. *Clin Radiol.* 1999;54:495–501.
35. Chabrol B, Decarie JC, Fortin G. The role of cranial MRI in identifying patients suffering from child abuse and presenting with unexplained neurological findings. *Child Abuse Negl.* 1999;23:217–228.
36. Barlow KM, Gibson RJ, McPhillips M, et al. Magnetic resonance imaging in acute nonaccidental head injury. *Acta Paediatr.* 1999;88:734–740.

37. Ewings-Cobbs L, Prasad M, Kramer L, et al. Acute neuroradiologic findings in young children with inflicted or noninflicted traumatic brain injury. *Childs Nerv Syst.* 2000;16:25–33.
38. Barnes PD, Robson CD. CT findings in hyperacute nonaccidental brain injury. *Pediatr Radiol.* 2000;30:74–81.
39. Slovis TL, Smith W, Kushner DC, et al. Imaging the child with suspected physical abuse. American College of Radiology. ACR Appropriateness Criteria. *Radiology.* 2000;215(suppl):805–809.
40. American Academy of Pediatrics. Section on radiology: diagnostic imaging of child abuse. *Pediatrics.* 2000;105:1345–1348.
41. Suh DY, Davis PC, Hopkins KL, et al. Nonaccidental pediatric head injury: diffusion-weighted imaging. *Neurosurgery.* 2001;49:309–320.
42. Biousse V, Suh DY, Newman NJ, et al. Diffusion-weighted MRI in shaken baby syndrome. *Am J Ophthalmol.* 2002;133:249–255.
43. Kemp AM. Investigating subdural haemorrhage in infants. *Arch Dis Child.* 2002;86:98–102.
44. Lonergan GF, Baker AM, Morey MK, et al. From the archives of the AFIP. Child abuse: radiologic-pathologic correlation. *Radiographics.* 2003;23:811–845.
45. Duhaime AC, Gennarelli TA, Thibault LE, et al. The shaken baby syndrome: a clinical, pathological, and biomechanical study. *J Neurosurg.* 1987;66:409–415.
46. Duhaime AC, Christian CW, Rorke LB, et al. Nonaccidental head injury in infants—the "shaken-baby syndrome". *N Engl J Med.* 1998;338:1822–1829.
47. Ommaya AK, Faas F, Yarnell P. Whiplash injury and brain damage: an experimental study. *JAMA.* 1968;204:285–289.
48. Guthkelch AN. Infantile subdural hematoma and its relationship to whiplash injuries. *BMJ.* 1971;2:430–431.
49. Uscinski R. Shaken baby syndrome: fundamental questions. *Br J Neurosurg.* 2002;16:217–219.
50. Ommaya A, Goldsmith W, Thibault L. Biomechanics and neuropathology of adult and paediatric head injury. *Br J Neurosurg.* 2002;16:220–242.
51. Prange MT, Coats B, Duhaime AC, et al. Anthropomorphic simulations of falls, shakes, and inflicted impacts in infants. *J Neurosurg.* 2003;99:143–150.
52. Goldsmith W, Plunkett J. Biomechanical analysis of the causes of traumatic brain injury in infants and children. *Am J Forensic Med Pathol.* 2004;25:89–100.
53. Bandak FA. Shaken baby syndrome: a biomechanics analysis of injury mechanisms. *Forensic Sci Int.* 2005;151:71–79.
54. Bandak FA. Shaken baby syndrome: a biomechanics analysis of injury mechanisms [author reply]. *Forensic Sci Int.* 2006;164:282–283.
55. Bertocci GE, Pierce MC, Deemer E, et al. Using test dummy experiments to investigate pediatric injury risk in simulated short-distanced falls. *Arch Pediatr Adolesc Med.* 2003;157:480–486.
56. Cory CZ, Jones MD, James DS, et al. The potential and limitations of utilizing head impact injury models to assess the likelihood of significant head injury in infants after a fall. *Forensic Sci Int.* 2001;123:89–106.
57. Bonnier C, Mesples B, Carpentier S, et al. Delayed white matter injury in a murine model of shaken baby syndrome. *Brain Pathol.* 2002;12:320–328.
58. Wolfson DR, McNally DS, Clifford MJ, et al. Rigid-body modeling of shaken baby syndrome. *Proc Inst Mech Eng [H].* 2005;219:63–70.
59. Raghupathi R, Margulies SS. Traumatic axonal injury after closed head injury in the neonatal pig. *J Neurotrauma.* 2002;19:843–845.
60. Raghupathi R, Mehr MF, Helfaer MA, et al. Traumatic axonal injury is exacerbated following repetitive closed head injury in the neonatal pig. *J Neurotrauma.* 2004;21:307–306.
61. Duhaime AC, Margulies SS, Durham SR. Maturation-dependent response of the piglet brain to scaled cortical impact. *J Neurosurg.* 2000;93:455–462.
62. Leestma JE. Case analysis of brain injured admittedly shaken infants, 54 cases 1969–2001. *Am J Forensic Med Pathol.* 2005;26:199–212.
63. Hwang SK, Kim SL. Infantile head injury, with special reference to the development of chronic subdural hematoma. *Childs Nerv Syst.* 2000;16:590–594.
64. Fung EL, Sung RY, Nelson EA, et al. Unexplained subdural hematoma in young children: is it always child abuse? *Pediatr Int.* 2002;44:37–42.
65. Dyer O. Brain haemorrhage in babies may not indicate violent abuse. *BMJ.* 2003;326:616.
66. Mackey M. After the Court of Appeal: R v Harris and others [2005] EWCA crim 1980. *Arch Dis Child.* 2006;91:873–875.
67. Gardner HB. Correlation between retinal abnormalities and intracranial abnormalities in the shaken baby syndrome. *Am J Ophthalmol.* 2003;135:745–746.
68. Miller M, Leestma J, Barnes P, et al. A sojourn in the abyss: hypothesis, theory, and established truth in infant head injury. *Pediatrics.* 2004;114:326.
69. Greenes DS, Schutzman SA. Clinical indicators of intracranial injury in head-injured infants. *Pediatrics.* 1999;104:861–867.
70. Greenes DS, Schutzman SA. Clinical significance of scalp abnormalities in asymptomatic head-injured infants. *Pediatr Emerg Care.* 2001;17:88–92.
71. Greenes DS, Schutzman SA. Occult intracranial injury in infants. *Ann Emerg Med.* 1998;32:680–686.
72. Gruskin KD, Schutzman SA. Head trauma in children younger than 2 years: are there predictors for complications? *Arch Pediatr Adolesc Med.* 1999;153:15–20.
73. Aoki N, Masuzawa H. Infantile acute subdural hematoma: clinical analysis of 26 cases. *J Neurosurg.* 1984;61:273–280.
74. Howard MA, Bell BA, Uttley D. The pathophysiology of infant subdural haematomas. *Br J Neurosurg.* 1993;7:355–365.
75. Parent AD. Pediatric chronic subdural hematoma: a retrospective comparative analysis. *Pediatr Neurosurg.* 1992;18:266–271.
76. Kawakami Y, Chikama M, Tamiya T, et al. Coagulation and fibrinolysis in chronic subdural hematoma. *Neurosurgery.* 1998;25:25–29.
77. Hwang SK, Kim SL. Infantile head injury, with special reference to the development of chronic subdural hematoma. *Childs Nerv Syst.* 2000;16:590–594.
78. Kim KA, Wang MY, Griffith PM, et al. Analysis of pediatric head injury from falls. *Neurosurg Focus.* 2000;8:1–9.
79. Piatt JH Jr. A pitfall in the diagnosis of child abuse: external hydrocephalus, subdural hematoma, and retinal hemorrhages. *Neurosurg Focus.* 1999;7:1–8.
80. Papasian N, Frim D. A theoretical model of benign external hydrocephalus that predicts a predisposition towards extra-axial hemorrhage after minor head trauma. *Pediatr Neurosurg.* 2000;33:188–193.
81. Pittman T. Significance of subdural hematoma in a child with external hydrocephalus. *Pediatr Neurosurg.* 2003;39:57–59.
82. McNeely PD, Atkinson JD, Saigal G, et al. Subdural hematomas in infants with benign enlargement of the subarachnoid spaces are not pathognomonic for child abuse. *Am J Neuroradiol.* 2006;27:1725–1728.
83. Ravid S, Maytal J. External hydrocephalus: a probable cause for subdural hematoma in infancy. *Pediatr Neurol.* 2003;28:139–141.
84. Plunkett J. Fatal pediatric head injuries caused by short-distance falls. *Am J Forensic Med Pathol.* 2001;22:1–12.
85. Stein S, Spettell C. Delayed and progressive brain injury in children and adolescents with head trauma. *Pediatr Neurosurg.* 1995;23,299–304.
86. Greenes D, Schutzman S. Occult intracranial trauma in infants. *Ann Emerg Med.* 1998;32:680–686.
87. Arbogast K, Margulies S, Christian C. Initial neurologic presentation in young children sustaining inflicted and unintentional fatal head injuries. *Pediatrics.* 2005;116:180–184.
88. Denton S, Mileusnic D. Delayed sudden death in an infant following an accidental fall: case report with review of the literature. *Am J Forensic Med Pathol.* 2003;24:371–376.
89. Bruce DA. Head injuries in the pediatric population. *Curr Probl Pediatr.* 1990;20:61–107.
90. Snoek JW, Minderhound JM, Wilmink JT. Delayed deterioration following mild head injury in children. *Brain.* 1984;107(Pt 1):15–36.
91. Kors EE, Terwindt GM, Vermeulen FL, et al. Delayed cerebral edema and fatal coma after minor head trauma: role of the CACNA1A calcium channel subunit gene and relationship with familial hemiplegic migraine. *Ann Neurol.* 2001;49:753–760.
92. Thiessen ML, Wolridge DP. Pediatric minor closed head injury. *Pediatr Clin North Am.* 2006;53:1–26.

93. Bruce DA. Delayed deterioration of consciousness after trivial head injury in childhood. *Br Med J (Clin Res Ed)*. 1984;289:715–716.
94. Chadwick DL, Chin S, Salerno C, et al. Deaths from falls in children: how far is fatal? *J Trauma*. 1991;31:1335.
95. Bruce DA, Alavi A, Bilaniuk L, et al. Diffuse cerebral swelling following head injuries in children: the syndrome of "malignant brain edema." *J Neurosurg*. 1981;54:170–178.
96. Poskitt K, Singhal A. Hyperacute cerebral edema in accidental pediatric head injury. Paper presented at: 44th Annual Meeting of the American Society of Neuroradiology/American Society of Pediatric Neuroradiology; May 2, 2006; San Diego, CA.
97. Schutzman SA, Barnes P, Duhaime AC, et al. Evaluation and management of children younger than two years old with apparently minor head trauma: proposed guidelines. *Pediatrics*. 2001;107: 983–993.
98. Schutzman SA, Greenes DS. Pediatric minor head trauma. *Ann Emerg Med*. 2001;37:65–74.
99. Reiber GD. Fatal falls in childhood: how far must children fall to sustain fatal head injuries? Report of cases and review of the literature. *Am J Forensic Med Pathol*. 1993;14:201–207.
100. Hall JR, Reyes HM, Horvat M, et al. The mortality of childhood falls. *J Trauma*. 1989;29:1273–1275.
101. Chiaviello CT, Christoph RA, Bond GR. Stairway-related injuries in children. *Pediatrics*. 1994;94:679–681.
102. Aldrich EF, Eisenberg HM, Saydjari C, et al. Diffuse brain swelling in severely head-injured children. A report from the NIH Traumatic Coma Data Bank. *J Neurosurg*. 1992;76:450–454.
103. Dashti SR, Decker DD, Razzap A, et al. Current patterns of inflicted head injury in children. *Pediatr Neurosurg*. 1999;31:302–306.
104. Geddes JF, Whitwell HL, Graham DI. Traumatic axonal injury: practical issues for diagnosis in medicolegal cases. *Neuropathol Appl Neurobiol*. 2000;26:105–116.
105. Geddes JF, Hackshaw AK, Vowles GH, et al. Neuropathology of inflicted head injury in children, I: pattern of brain injury. *Brain*. 2001;124:1290–1298.
106. Geddes JF, Hackshaw AK, Vowles GH, et al. Neuropathology of inflicted head injury in children, II: microscopic brain injury in infants. *Brain*. 2001;124:1299–1306.
107. Geddes JF, Tasker RC, Hackshaw AK, et al. Dural haemorrhage in non-traumatic infant deaths: does it explain the bleeding in "shaken baby syndrome?" *Neuropathol Appl Neurobiol*. 2003;29:14–22.
108. Geddes JF, Whitwell HL. Inflicted head injury in infants. *Forensic Sci Int*. 2004;146:83–88.
109. Geddes J. Pediatric head injury. In: Golden JA, Harding BN, eds. *Developmental Neuropathology*. Basel, Switzerland: ISN Neuropath Press; 2004:chap 23.
110. Lantz PE, Sinal SH, Stanton CA, et al. Evidence based case report: perimacular retinal folds from childhood head trauma. *BMJ*. 2004;328;754–756.
111. Aryan HE, Ghosheh FR, Jandial R, et al. Retinal hemorrhage and pediatric brain injury: etiology and review of the literature. *J Clin Neurosci*. 2005;12:624–631.
112. Gilliland MGF. Why do histology on retinal haemorrhages in suspected non-accidental injury. *Histopathology*. 2003;43:592–602.
113. Christian CW, Taylor AA, Hertle RW, et al. Retinal hemorrhages caused by accidental household trauma. *J Pediatr*. 1999;135:125–127.
114. Barnes PD. Imaging of the central nervous system (CNS) in suspected or alleged non-accidental injury (NAI). *Gyrations* [official newsletter of the American Society of Pediatric Neuroradiology]. 2007;2:5–7.
115. Hymel KP, Jenny C, Block RW. Intracranial hemorrhage and rebleeding in suspected victims of abusive head trauma: addressing the forensic controversies. *Child Maltreat*. 2002;7:329–348.
116. Tung GA, Kumar M, Richardson RC, et al. Comparison of accidental and nonaccidental traumatic head injury in children on noncontrast computed tomography. *Pediatrics*. 2006;118:626–633.
117. Vinchon M, Noule N, Tchofo PJ, et al. Imaging of head injuries in infants: temporal correlates and forensic implication for the diagnosis of child abuse. *J Neurosurg*. 2004;101:44–52.
118. Wells R, Sty J. Traumatic low attenuation subdural fluid collections in children younger than 3 years. *Arch Pediatr Adolesc Med*. 2003;157:1005–1010.
119. Wolpert S, Barnes P. *MRI in Pediatric Neuroradiology*. New York, NY: Mosby Year Book; 1992.
120. Bradley WG Jr. MR appearance of hemorrhage in the brain. *Radiology*. 1993;189:15–26.
121. Zuerrer M, Martin E, Boltshauser E. MR imaging of intracranial hemorrhage in neonates and infants at 2.35 tesla. *Neuroradiology*. 1991;33:223–229.
122. Duhaime AC, Christian C, Armonda R, et al. Disappearing subdural hematomas in children. *Pediatr Neurosurg*. 1996;25:116–122.
123. Barkovich A. *Pediatric Neuroimaging*. Philadelphia, PA: Lippincott-Raven; 2005:190–290.
124. Winkler P, Zimmerman RA. Perinatal brain injury. In: Zimmerman RA, Gibby WA, Carmody RF, eds. *Neuroimaging: Clinical and Physical Principles*. New York, NY: Springer; 2000:531–583.
125. Barnes PD. Neuroimaging and the timing of fetal and neonatal brain injury. *J Perinatol*. 2001;21:44–60.
126. Blankenburg F, Barnes P. Structural and functional imaging of hypoxic-ischemic injury (HII) in the fetal and neonatal brain. In: Stevenson D, Benitz W, Sunshine P, eds. *Fetal and Neonatal Brain Injury*. 3rd ed. New York, NY: Cambridge University Press; 2003.
127. Fullerton HJ, Johnston SC, Smith WS. Arterial dissection and stroke in children. *Neurology*. 2001;57:1155–1160.
128. Stiefel D, Eich G, Sacher P. Posttraumatic dural sinus thrombosis in children. *Eur J Pediatr Surg*. 2000;10:41–44.
129. Pang D, Wilberger JE. Spinal cord injury without radiographic abnormality in children—the SCIWORA syndrome. *J Trauma*. 1918;29:654–664.
130. Cirak B, Ziegfeld S, Knight VM, et al. Spinal injuries in children. *J Pediatr Surg*. 2004;39:602–612.
131. Brown RL, Brunn MA, Garcia VF. Cervical spine injuries in children. *J Pediatr Surg*. 2001;36:1107–1114.
132. Geddes JF, Talbert DG. Paroxysmal coughing, subdural and retinal bleeding: a computer modeling approach. *Neuropathol Appl Neurobiol*. 2006;32:625–634.
133. Sirotnak A. Medical disorders that mimic abusive head trauma. In: Frasier L, Farley KR, Alexander R, et al, eds. *Abusive Head Trauma in Infants and Children: A Medical, Legal, and Forensic Reference*. St Louis, MO: GW Medical Publishing; 2006:191–226.
134. Talbert DG. The "sutured skull" and intracranial bleeding in infants. *Med Hypotheses*. 2006;66:691–694.
135. Ganesh A, Jenny C, Geyer J, et al. Retinal hemorrhages in type I osteogenesis imperfecta after minor trauma. *Ophthalmology*. 2004;111:1428–1431.
136. Marlow A, Pepin M, Byers P. Testing for osteogenesis imperfecta in cases of suspected non-accidental injury. *J Med Genet*. 2002;39: 382–386.
137. Clemetson CAB. Caffey revisited: a commentary on the origin of "shaken baby syndrome." *J Am Phys Surg*. 2006;11:20–21.
138. Clemetson CAB. Is it "shaken baby," or Barlow's disease variant? *J Am Phys Surg*. 2004;9:78–80.
139. Innis MD. Vaccines, apparent life-threatening events, Barlow's disease, and questions about "shaken baby syndrome." *J Am Phys Surg*. 2006;11:17–19.
140. American Academy of Pediatrics Committee on Fetus and Newborn. Controversies concerning vitamin K and the newborn. *Pediatrics*. 2003;112(1 Pt 1):191–192.
141. Vermeer C, Knapen MHJ, Schurgers J. Vitamin K and metabolic bone disease. *J Clin Pathol*. 1998;51:424–426.
142. Rutty GN, Smith CM, Malia RG. Late-form hemorrhagic disease of the newborn: a fatal case report with illustration of investigations that may assist in avoiding the mistaken diagnosis of child abuse. *Am J Forensic Med Pathol*. 1999;20:48–51.
143. Brousseau TJ, Kissoon N, McIntosh B. Vitamin K deficiency mimicking child abuse. *J Emerg Med*. 2005;29:283–288.
144. Ziegler EE, Hollis BW, Nelson SE, et al. Vitamin D deficiency in breastfed infants in Iowa. *Pediatrics*. 2006;118:603–610.
145. Hayashi T, Hashimoto T, Fukuda S, et al. Neonatal subdural hematoma secondary to birth injury. *Childs Nerv Syst*. 1987;3:23–29.
146. Durham SR, Duhaime AC. Maturation-dependent response of the immature brain to experimental subdural hematoma. *J Neurotrauma*. 2007;24:5–14.

147. Ney JP, Joseph KR, Mitchell MH. Late subdural hygromas from birth trauma. *Neurology*. 2005;65:517.
148. Chamnanvanakij S, Rollins N, Perlman JM. Subdural hematoma in term infants. *Pediatr Neurol*. 2002;26:301–304.
149. Hadzikaric N, Al-Habib H, Al-Ahmad I. Idiopathic chronic subdural hematoma in the newborn. *Childs Nerv Syst*. 2006;22: 740–742.
150. Whitby EH, Griffiths PD, Rutter S, et al. Frequency and natural history of subdural haemorrhages in babies and relation to obstetric factors. *Lancet*. 2004;363:846–851.
151. Looney CB, Smith JK, Merck LH, et al. Intracranial hemorrhage in asymptomatic neonates: prevalence on MRI and relationship to obstetric and neonatal risk factors. *Radiology*. 2007;242:535–541.
152. Volpe JJ. *Neurology of the Newborn*. 4th ed. Philadelphia, PA: Saunders; 2000.
153. Burrows P, Robertson R, Barnes P. Angiography and the evaluation of cerebrovascular disease in childhood. *Neuroimaging Clin N Am*. 1996;6:561–588.
154. Fordham LA, Chung CJ, Donnelly LF. Imaging of congenital vascular and lymphatic anomalies of the head and neck. *Neuroimaging Clin N Am*. 2000;10:117–136.
155. Rogers MA, Klug GL, Siu KH. Middle fossa arachnoid cysts in association with subdural haematomas. *Br J Neurosurg*. 1990;4:497–502.
156. Strauss KA, Puffenberger EG, Robinson DL, et al. Type I glutaric aciduria, part 1: natural history of 77 patients. *Am J Med Genet*. 2003;121:38–52.
157. Rooms L, Fitzgerald N, McClain KL. Hemophagocytic lymphohistiocytosis masquerading as child abuse. *Pediatrics*. 2003; 111:636–640.
158. Carvalho KS, Bodensteiner JB, Connolly PJ, et al. Cerebral venous thrombosis in children. *J Child Neurol*. 2001;16:574–585.
159. Fitzgerald KC, Williams LS, Garg BP, et al. Cerebral sinovenous thrombosis in the neonate. *Arch Neurol*. 2006;63:405–409.
160. DeVeber G, Andrew M, Adams C, et al. Canadian Pediatric Ischemic Stroke Study Group. Cerebral sinovenous thrombosis in children. *N Engl J Med*. 2001;345:417–423.
161. Barnes C, DeVeber G. Prothrombotic abnormalities in childhood ischaemic stroke. *Thromb Res*. 2006;118:67–74.
162. Kleinman P. *Diagnostic Imaging of Child Abuse*. 2nd ed. New York, NY: Mosby Year Book; 1998.
163. Miller ME. Hypothesis: fetal movement influences fetal and infant bone strength. *Med Hypotheses*. 2005;65:880–886.
164. Ablin DS, Sane SM. Non-accidental injury: confusion with temporary brittle bone disease and mild osteogenesis imperfecta. *Pediatr Radiol*. 1997;27:111–113.
165. Miller ME. Another perspective as to the cause of bone fractures in potential child abuse. *Pediatr Radiol*. 2000;30:495–496.
166. Grayev AM, Boal DK, Wallach DM, et al. Metaphyseal fractures mimicking abuse during treatment for clubfoot. *Pediatr Radiol*. 2001;31:55–563.
167. Chalumeau M, Foix-l'Helias L, Scheinmann P, et al. Rib fractures after chest physiotherapy for bronchiolitis or pneumonia in infants. *Pediatr Radiol*. 2002;32:644–647.
168. Miller ME. Infants at higher risk to fracture than the general population. *Pediatr Radiol*. 2003;33:733–734.
169. Miller ME. The bone disease of preterm birth: a biomechanical perspective. *Pediatr Res*. 2003;53:10–15.
170. Dabezies E, Warren PD. Fractures in very low birth weight infants with rickets. *Clin Orthop*. 1997;335:233–239.
171. Hartmann RW Jr. Radiological case of the month. Rib fractures produced by birth trauma. *Arch Pediatr Adolesc Med*. 1997;151:947–948.
172. Miller ME. The lesson of temporary brittle bone disease: all bones are not created equal. *Bone*. 2003;33:466–474.
173. Jenny C. Evaluating infants and young children with multiple fractures. *Pediatrics*. 2006;118:1299–1303.
174. Prosser I, Maguire S, Harrison SK, et al. How old is this fracture? Radiologic dating of fractures in children: a systematic review. *AJR Am J Roentgenol*. 2005;184:1282–1286.

EXHIBIT K

Legal Studies Research Paper Series Paper No. 1195

Houston Journal of Health and Policy (forthcoming)

SHAKEN BABY SYNDROME, ABUSIVE HEAD TRAUMA, AND ACTUAL INNOCENCE: GETTING IT RIGHT

Keith A. Findley, University of Wisconsin Law School

Patrick D. Barnes, Stanford School of Medicine

David A. Moran, University of Michigan Law School

Waney Squier, John Radcliffe Hospital, Oxford, England

SHAKEN BABY SYNDROME, ABUSIVE HEAD TRAUMA, AND ACTUAL INNOCENCE: GETTING IT RIGHT

Patrick D. Barnes*
Keith A. Findley†
David A. Moran‡
Waney Squier§

In the past decade, the existence of shaken baby syndrome (SBS) has been called into serious question by biomechanical studies, the medical and legal literature, and the media. As a result of these questions, SBS has been renamed abusive head trauma (AHT). This is, however, primarily a terminological shift: like SBS, AHT refers to the two-part hypothesis that one can reliably diagnose shaking or abuse from three internal findings (subdural hemorrhage, retinal hemorrhage and encephalopathy) and that one can identify the perpetrator based on the onset of symptoms. Over the past decade, we have learned that this hypothesis fits poorly with the anatomy and physiology of the infant brain, that there are many natural and accidental causes for these findings, and that the onset of symptoms does not reliably indicate timing.

In the last issue of this journal, Dr. Sandeep Narang marshaled the arguments and evidence that he believes support the diagnostic specificity of the medical signs that are used to diagnose SBS/AHT. Dr. Narang does not dispute the alternative diagnoses but nonetheless argues that, in the absence of a proven alternative, the SBS/AHT hypothesis is sufficiently reliable to support criminal convictions. The cited studies do not, however, support this position since they assume the validity of the hypothesis without examining it and classify cases accordingly, often without considering alternative diagnoses. To address this problem, Dr. Narang argues that, in diagnosing SBS/AHT, we should rely on the judgment of child abuse pediatricians and other clinicians who endorse the hypothesis. Reliance on groups that endorse a particular hypothesis is, however, antithetical to evidence-based medicine and Daubert, which require an objective assessment of the scientific evidence.

In the past decades, thousands of parents and caretakers have been accused – and many convicted – of abusing children based on a hypothesis that is not scientifically supported. While we must do everything in our power to protect children, we must refrain from invoking abuse as a default diagnosis for medical findings that are complex, poorly understood and have a wide range of causes, some doubtlessly yet unknown. To this end, we are calling for collaboration between the medical and legal communities for the sole purpose of "getting it right."

---

* Chief, Section of Pediatric Neuroradiology; Director, Pediatric MRI and CT, Lucile Packard Children's Hospital, Stanford University School of Medicine.
† Assistant Professor of Law, University of Wisconsin Law School; Co-Director, Wisconsin Innocence Project; President, The Innocence Network.
‡ Clinical Professor of Law, University of Michigan Law School; Co-Director, Michigan Innocence Clinic.
§ Paediatric Neuropathologist, John Radcliffe Hospital, Oxford, England.

TABLE OF CONTENTS

I. INTRODUCTION 1
II. FROM SBS TO AHT: A DIAGNOSIS IN FLUX 5
A. A Plethora of Terms 5
1. Shaking 6
2. Shaken baby syndrome (SBS) 6
3. Shaken impact syndrome 6
4. Abusive head trauma 6
5. Blunt force trauma 7
6. Semantic confusion and the courts 7
B. A Brief History of SBS/AHT 9
1. The origins 9
2. The warnings 12
3. 2001: a developing schism 13
4. A decade of debate 17
a. 2002 NIH conference 17
b. Biomechanics 19
c. SBS and evidence-based medicine 20
d. Alternative diagnoses 20
e. The position papers revisited 22
f. Increasing divergence 23
g. The triad: where are we now? 25
C. Ongoing Debates 25
1. Short falls 26
2. Timing ("lucid intervals") 29
3. Retinal hemorrhages 30
4. Bruises, fractures and other findings 33
5. Confessions 34
6. New hypotheses 38
III. THE MEDICAL EVIDENCE: OLD AND NEW 39
A. Literature Supporting the AHT Diagnosis 39
1. The methodology 40
2. The evidence 41
a. Duhaime (1992) 42
b. Ewing-Cobbs (1998) 43
c. Feldman (2001) 43
d. Wells (2002) 43
e. Bechtel (2004) 44
f. Hobbs (2005) 44
g. Vinchon (2005) 44
h. Matschke (2009) 45
i. Vinchon (2010) 45
j. Other studies 46
3. The Flaws 46
a. Circularity 46

b. Rule-out diagnoses....48
c. Clinical judgment....51
d. Observer bias....52
e. Reversing the burden of proof....54
f. Interpretive errors....55
i. P-values....55
ii. The Prosecutor's Fallacy....55
iii. Improper classifications....58
B. The Skeptics: New Research, Old Anatomy....58
1. Studies that identify the lack of support for the traditional SBS hypothesis....58
2. Studies that identify problems with the SBS/AHT hypothesis....60
3. A shifting paradigm....62
IV. MEDICAL AND LEGAL STANDARDS OF RELIABILITY....64
A. Medical Diagnosis: Art or Science?....64
B. Daubert: Is SBS/AHT Ready for the Courtroom?....67
C. The Costs of Misdiagnosis....71
V. THE PATH FORWARD....71
A. Research....72
B. Working Together....72
C. Acknowledging the Complexities....73
D. Working Under Conditions of Uncertainty....75

I. INTRODUCTION

For decades, shaken baby syndrome (SBS) was an accepted medical and legal diagnosis. As the shaking mechanism came into serious question, SBS was renamed abusive head trauma (AHT). Regardless of terminology, SBS/AHT refers to the two-part medicolegal hypothesis that, in the absence of a confirmed alternative explanation, one can reliably diagnose shaking or abuse from three internal findings – subdural hemorrhage, retinal hemorrhage and encephalopathy (brain abnormalities and/or neurological symptoms), and that one can identify the perpetrator based on the onset of symptoms. Because the consequences of an SBS/AHT diagnosis can devastate children and families, it is critical to assess the reliability of the diagnosis under the standards of evidence-based medicine[5] and *Daubert v. Merrell Dow Pharmaceuticals, Inc.*[6] Dr. Sandeep Narang's article in this journal identifies the research basis for the SBS/AHT hypothesis and the applicable medicolegal standards.[7] However, in concluding that the SBS/AHT hypothesis meets the standards of evidence-based medicine and *Daubert,* the article neglects the underlying flaws in the supporting research and the shift in our understanding of the science over the past decade.

For all the heat in the debates about the validity of SBS/AHT, there is in reality a growing, if frequently unexpressed, consensus on the nature of the problem and the flaws in the hypothesis. Today, there is general agreement that child abuse was historically under-recognized and that abuse can produce subdural hemorrhage, retinal hemorrhage and brain damage – the "triad" of medical findings that has traditionally been used to confirm shaking or other forms of abuse.[8] There is also general agreement that violently shaking a child is unacceptable and could cause serious injury or even death.[9] At the same time, there is now

---

[5] *See, e.g.,* Connie Schardt & Jill Mayer, *Introduction to Evidence-Based Medicine,* Duke University Medical Center Library and Health Sciences Library, UNC-Chapel Hill (2010) at http://www.hsl.unc.edu/services/tutorials/ebm/index.htm; Gordon H. Guyatt et al., *Users' Guides to the Medical Literature XXV, Evidence-Based Medicine: Principles for Applying the Users' Guides to Patient Care*, 10 J. AM. MED. ASS. 1290 (2000).

[6] 509 U.S. 579 (1993).

[7] Sandeep K. Narang, *A Daubert Analysis of Abusive Head Trauma/Shaken Baby Syndrome*, 11 HOUS. J. HEALTH L. & POL'Y 505, 506, 507, 538-559 (2011).

[8] *See, e.g., id.*, at 522, 523, 569.

[9] *See, e.g.,* Emily Bazelon, Mary Case, Christopher Greeley, Ronald H. Uscinski, Waney Squier, Round Table Discussion: Anatomy of an AHT Diagnosis, Investigation and Prosecution, New York City Abusive Head Trauma/Shaken Baby Syndrome Training Conference (Sept. 23, 2011) (notes on file with authors) (all participants agreed that violent shaking is dangerous and may injure or kill an infant); Kay Rauth-Farley, Lori D. Frasier, Robert N. Parrish, *Current Perspectives on Abusive Head Trauma*, ABUSIVE HEAD TRAUMA IN INFANTS AND CHILDREN, A MEDICAL, LEGAL, AND FORENSIC REFERENCE 1, 1 (2006) ("It is widely accepted that shaking a young child or infant is dangerous").

widespread, if not universal, agreement that the presence of the triad alone – or its individual components – is not enough to diagnose abuse. In the United Kingdom, the Crown Prosecution Service Guidelines of March 2011 endorsed this view,[10] while in the U.S. the diagnostic specificity of the "triad" was recently described as a "myth" by a leading proponent of the SBS/AHT hypothesis.[11] As we develop more fully below, there is also a growing consensus that certain features of the diagnosis were inaccurate, including some that were frequently used to obtain criminal convictions. For example, it is no longer generally accepted that short falls can never cause the triad, that there can be no period of lucidity between injury and collapse (a key element in identifying the perpetrator), or that massive force – typically described as the equivalent of a multi-story fall or car accident – is required.[12]

As Dr. Narang points out, the list of alternative causes for the triad or its components is now so broad that it cannot be addressed in a single article.[13] One of the child abuse textbooks recommended by Dr. Narang lists the differential diagnosis (alternative causes or "mimics") as accidental causes; prenatal and perinatal conditions, including birth trauma; congenital malformations; genetic conditions; metabolic disorders; coagulation disorders, including venous sinus thrombosis (a form of childhood stroke); infectious disease; vasculitis; autoimmune conditions; oncology; toxins and poisons; nutritional deficiencies; and complications from medical-surgical procedures, including lumbar puncture.[14] In all likelihood, other causes are still undiscovered.[15] Like

---

[10] Crown Prosecution Service, *Non Accidental Head Injury Cases (NAHI, formerly referred to as Shaken Baby Syndrome [SBS]) - Prosecution Approach*, March 24, 2011 ("it is unlikely that a charge for a homicide (or attempted murder or assault) offence could be justified where the only evidence available is the triad of pathological features"), available at http://www.cps.gov.uk/legal/l_to_o/non_accidental_head_injury_cases/.

[11] Carole Jenny, Presentation, *The Mechanics: Distinguishing AHT/SBS from Accidents and Other Medical Conditions*, slide 11, New York City Abusive Head Trauma/Shaken Baby Syndrome Training Conference (Sept. 23, 2011) at http://www.queensda.org/SBS_Conference/SBC2011.html.

[12] *See infra* notes 111, 124, 125, 220 and accompanying text.

[13] Narang, *supra* note 3, at 507, note 13 ("A thorough examination of the literature behind all the possible injuries and all potential causes (short falls, biomechanics of head injury, etc.) is simply too broad and beyond the scope of this paper"). *See also* Narang, *supra* note 3, at Appendices B and C (differential diagnosis for subdural hemorrhage includes inflicted trauma, accidental trauma, birth trauma, metabolic disease, nutritional deficiencies, genetic syndromes, clotting disorders, tumors and infection; differential diagnosis for retinal hemorrhages include all of the above as well as anemia, vasculitis, hypoxia, hypotension, hypertension and increased intracranial pressure); Julian T. Hoff et al., *Brain Edema*, 22 NEUROSURG. FOCUS 1 (2007) (causes of brain edema include trauma, stroke and tumors).

[14] Andrew P. Sirotnak, *Medical Disorders that Mimic Abusive Head Trauma, in* ABUSIVE HEAD TRAUMA IN INFANTS AND CHILDREN, A MEDICAL, LEGAL, AND FORENSIC REFERENCE 191, 193-214 (2006); M. Denise Dowd, *Epidemiology of Traumatic Brain Injury: Recognizing Unintentional Head Injuries in Children, in*

Dr. Narang, we refer the reader to the literature for a discussion of the alternative causes.[16]

Given this emerging consensus, our disagreement with Dr. Narang is narrow but critical. Since biomechanical studies have consistently concluded that shaking does not generate enough force to produce the types of traumatic damage associated with SBS/AHT, particularly in the absence of neck damage, Dr. Narang does not defend shaking as a mechanism or argue that there are no diagnostic alternatives. Instead, as is typical in the current debates about these issues, he contends that the less-specific diagnosis of AHT is supported by current medical science when subdural and retinal hemorrhage are identified and other known causes ruled out.[17]

Changing the name of the syndrome from SBS to AHT does not, however, resolve the disagreement. In describing AHT, Dr. Narang does not offer new evidence but instead relies on the assumptions that provided the basis for the SBS hypothesis.[18] This hypothesis assumed that each element of the triad was, virtually by definition, traumatic, *i.e.*, that subdural and retinal hemorrhages were caused by the traumatic rupture of bridging veins and retinal blood vessels and that encephalopathy was caused by the traumatic rupture of axons (the nerve fibers that connect the cells throughout the brain). We now know, however, that the triad does not necessarily or generally reflect the traumatic rupture of bridging veins or retinal blood vessels; that the encephalopathy virtually always reflects hypoxia-ischemia (lack of oxygen) rather than the traumatic tearing of axons; and that the triad can

---

ABUSIVE HEAD TRAUMA IN INFANTS AND CHILDREN, A MEDICAL, LEGAL, AND FORENSIC REFERENCE 11, 12-14 (2006).

[15] We are, for example, just beginning to identify the many variations of the human genome, the thousands of metabolytes and enzymes that must function properly to sustain life, and the unique anatomic and physiological characteristics of the infant brain.

[16] In 2011, two of the co-authors of this article – Dr. Barnes and Dr. Squier – addressed the differential diagnoses in major invited reviews of the medical evidence on SBS/AHT in the fields of pediatric neuroradiology and pediatric neuropathology, their own specialties. Patrick D. Barnes, *Imaging of Nonaccidental Injury and the Mimics: Issues and Controversies in the Era of Evidence-Based Medicine*, 49 RADIOL. CLIN. N. AM. 205 (2011); Waney Squier, *The "Shaken Baby" Syndrome: Pathology and Mechanisms*, 122 ACTA NEUROPATHOL. 519 (2011). For a more complete discussion of the literature, we refer the readers to these reviews and to the articles cited by Dr. Narang.

[17] Narang, *supra* note 3, at 569.

[18] In describing AHT causation, Dr. Narang relies upon the classic SBS hypothesis, with no reference to the more recent literature (discussed below). *See, e.g., id.* at 541 ("In inertial [i.e. shaking] events, the acceleration-deceleration motion of the brain results in strain upon the cortical bridging veins which exceeds their tolerance levels and subsequently leads to rupture and hemorrhage (subdural and/or subarachnoid)) and 552-553 ("[S]everal lines of research and analysis point towards acceleration-deceleration forces at the vitreo-retinal interface…as the causative mechanism for severe [retinal hemorrhages]").

also result from natural disease processes and accidents. Consequently, it is no longer valid to reason backwards from the triad to a diagnosis of trauma or abuse.

The AHT label also raises new problems. Without an identified mechanism, it is not possible for biomechanical engineers to reconstruct or for doctors, judges or juries to critically evaluate the proposed mechanism or mechanisms. The AHT label does not, moreover, address the more recent criticisms of SBS/AHT, which have shifted from biomechanics to the unique characteristics of the developing brain. Finally, like the SBS label, the AHT label subsumes the answer to the question "what causes the triad or its elements" within its very name, making it difficult to discuss the issues objectively.

Since the existing evidence does not meet the standards of evidence-based medicine and we cannot ethically experiment with babies, Dr. Narang suggests that we rely on the "clinical judgment" of the doctors, particularly child abuse pediatricians, who endorse the SBS/AHT hypothesis and defer to the literature that assumes the accuracy of their judgments.[19] As a practical matter, this would shield the SBS/AHT hypothesis from the scientific scrutiny envisioned by evidence-based medicine and *Daubert* and eliminate any claim that the hypothesis has been scientifically validated. We suggest that this approach also violates the medical and legal precepts of "first do no harm" and "innocent until proven guilty."

While child abuse that results in neurological damage or death is horrific, particularly when committed by parents and caretakers who literally hold in their hands the lives of their infants, we have learned from the daycare cases of the 1980s and 1990s that the strong emotions that accompany allegations of child abuse can increase the likelihood of false convictions.[20] In a 1990 symposium on pretrial publicity, Judge Abner Mivka, a highly respected member of the U.S. Court of Appeals for the District of Columbia, observed:

---

[19] Narang *supra* note 3, at 579-580 (arguing that the relevant scientific community be limited to those who have obtained subspecialty certification or are eligible for subspecialty certification in the field of child abuse pediatrics). This certification program, which was created by leading advocates of the SBS/AHT hypothesis, incorporates the traditional SBS/AHT hypothesis into its curriculum. *See* American Board of Pediatrics Subboard of Child Abuse Pediatrics, *Child Abuse Pediatrics Content Outline,* (January 2009) (on file with authors); Robert W. Block & Vincent J. Palusci, *Child Abuse Pediatrics: A New Pediatric Subspecialty*, 148 J. OF PEDIATRICS 711 (2006).

[20] *See, e.g.,* DOROTHY RABINOWITZ, NO CRUELER TYRANNIES (1st ed. 2003) (reporting on daycare, Wenatchee and other child sex abuse scandals of the 1980s and 1990s); Maggie Jones, *Who Was Abused?,* N. Y. TIMES, Sept. 19, 2004, at http://query..com/gst/fullpage.html?res=9F03EFD61330F93AA2575AC0A9629C8B63&scp=1&sq=maggie jones who was abused&st=cse&pagewanted=1 (reporting on Bakersfield scandals); *Summary of the Cleveland Inquiry,* 297 BRIT. MED. J. 190 (1988).

> I do not think that you can get a fair child abuse trial before a jury anywhere in the country. I really don't . . . I don't care how sophisticated or smart jurors are, when they hear that a child has been abused, a piece of their mind closes up and this goes for the judge, the juror, and all of us.[21]

Given these dangers, it is critical to carefully assess the quality of the evidence used to diagnose child abuse and to make clear the extent to which the diagnosis rests on hypotheses or personal opinion rather than scientific knowledge. This is particularly important when judges and jurors are being asked to render judgments on unresolved and highly controversial issues in complex areas of medicine.

In Part II, we briefly review the changes in the SBS/AHT hypothesis over the past decade and identify the issues that are currently the subject of debate. The shifts can be captured in a sentence: since 2000, we have learned that much of what we thought we knew was wrong. In Part III, we examine the quality of the research that Dr. Narang cites to support the SBS/AHT hypothesis as well as the research that casts doubt on this hypothesis. In Part IV, we apply the applicable medical and legal standards to this research. In Part V, we suggest a path forward to help us better differentiate between child abuse and the wide array of accidental and natural causes that may produce the same or similar findings. It is our hope that Dr. Narang and others will join us in this endeavor to "get it right."

II. FROM SBS TO AHT: A DIAGNOSIS IN FLUX

Our increased understanding of the infant brain and the biomechanics of injury is reflected in an evolving terminology that acknowledges the flaws in the original SBS hypothesis. Despite widespread acknowledgement of these flaws, the new terminology (AHT) retains the automatic diagnosis of abuse for the medical findings previously attributed to shaking and rests on the same assumptions as SBS, many of which have been discredited or disproven. After clarifying the terminology, we discuss the shifts in the literature that resulted in the new terminology. We then identify the areas of current agreement and debate.

A. A Plethora of Terms

In addressing the changes in the SBS/AHT hypothesis, it is important to distinguish between five terms and diagnoses: "shaking," "shaken baby syndrome," "shaken impact syndrome," "abusive head

[21] Forum, *Panel One: What Empirical Research Tells Us, and What We Need to Know About Juries and the Quest for Impartiality*, 40 AM. U. L. REV. 547, 564-565 (1991).

trauma" and "blunt force trauma." Much of the disagreement in this area reflects the confusion of these terms and conflation of the underlying concepts.

1. ***Shaking.*** "Shaking" refers to the physical act of shaking a child, irrespective of injury. Shaking to punish or in frustration is always inappropriate. In infants with large heads and weak necks – or even in older children – violent shaking may lead to disastrous consequences, particularly in a child with predisposing factors.

2. ***Shaken baby syndrome.*** "Shaken baby syndrome" (SBS) refers to hypothesis that violent shaking may be reliably diagnosed based on the triad of subdural hemorrhage, retinal hemorrhage, and encephalopathy (brain damage) if the caretakers do not describe a major trauma (typically described as equivalent to a motor vehicle accident or fall from a multistory building) and no alternative medical explanation is identified. Under this hypothesis, the rapid acceleration and deceleration of shaking causes movement of the brain within the skull, resulting in the traumatic rupture of bridging veins, retinal blood vessels and nerve fibers throughout the brain (diffuse axonal injury). This hypothesis came into question when biomechanical studies consistently concluded that shaking generated far less force than impact, did not meet established injury thresholds, and would be expected to injure the neck before causing bridging vein rupture or diffuse axonal injury.

3. ***Shaken impact syndrome.*** "Shaken impact syndrome" was advanced to address the biomechanical criticisms of shaking as a causal mechanism for the triad. Under this hypothesis, subdural hemorrhage, retinal hemorrhage and encephalopathy were attributed to shaking followed by impact, such as tossing or slamming the child onto a hard or soft surface. If there were no bruises or other signs of impact, it was hypothesized that the child was thrown onto a soft surface, such as a mattress or pillow.

4. ***Abusive head trauma.*** As shaking came under increasing scrutiny, a plethora of new terms arose that did not invoke shaking as a mechanism.[22] At present, the most popular replacement term – and the term used by Dr. Narang – is abusive head trauma, or AHT. AHT refers to any deliberately inflicted injury to the head, regardless of mechanism. In 2009, the American Academy of Pediatrics recommended that pediatricians use this term instead of SBS but endorsed shaking as a plausible mechanism based on confession evidence.[23] AHT also includes hitting the child on the head, crushing the child, throwing the child onto a hard or soft surface, or any other conceivable manner of

[22] These terms include "intentional traumatic brain injury (iTBI)," "nonaccidental injury (NAI)," "nonaccidental head injury (NAHI)," "nonaccidental trauma (NAT)," "inflicted neurotrauma" and "abusive head trauma (AHT)".

[23] Cindy W. Christian, Robert Block and the Committee on Child Abuse and Neglect, *Abusive Head Trauma in Infants and Children*, 123 PEDIATRICS 1409 (2009).

harming the head. Under the AHT hypothesis, such acts may be inferred from the triad of findings previously attributed to shaking, with or without other evidence of trauma, at least in the absence of another acceptable explanation. Used in this sense, AHT is most often used by pediatricians.

5. ***Blunt force trauma.*** Blunt force trauma to the head refers to any impact that does not penetrate the scalp, including accidents (e.g., falls onto the floor or other surfaces) and abuse (e.g., hitting the child on the head or throwing the child on the floor). This term does not imply intent and is used in cases with skull fractures and bruises as well as in cases that rely primarily or exclusively on the triad. This term is most often used by forensic pathologists.

6. ***Semantics and the courts.*** As reflected in Dr. Narang's article, the trend in recent years has been to move away from terms involving shaking towards generalized terms such as AHT, which avoids the criticisms of shaking by relying upon an undetermined mechanism. Without a defined mechanism, however, it is difficult for parents or caretakers to defend themselves: how does one defend against an unknown mechanism, particularly one that leaves no clues as to its cause? In effect, by changing the name, supporters of the AHT hypothesis continue to rely on traditional SBS assumptions – specifically, the assumption that the triad findings are caused largely or entirely by trauma – while discarding the shaking mechanism, producing what may be viewed as a medicolegal "bait and switch."

When combined with unfamiliar medical concepts, these terminological shifts can result in considerable confusion, even at the level of the U.S. Supreme Court. This confusion is exemplified by the U.S. Supreme Court decision in *Cavazos v. Smith.*[24] In *Smith,* a California grandmother with no history of abuse or neglect was convicted of causing the death of her 7-week-old grandson by violent shaking. This was not a classic SBS/AHT case since the child had minimal subdural/subarachnoid hemorrhage with no retinal hemorrhage or brain swelling. There were no fractures, no sprains and no other indicia of trauma other than a "tiny" abrasion and corresponding bruise, which the prosecution's medical expert agreed did not produce brain trauma.[25] The state's experts testified nonetheless that the death was consistent with violent shaking that caused the brain or brainstem – not just the bridging veins and axons – to tear in vital areas.[26] The Ninth Circuit overturned the conviction, stating that there was "no physical evidence of . . . tearing or shearing, and no other evidence supporting death by violent shaking."[27] A 6-3 majority of the Supreme Court reversed the Ninth Circuit, stating that the Ninth Circuit's assertion that

[24] Cavazos v. Smith, 132 S. Ct. 2 (2011) (per curiam).
[25] *Id.* at 3 (Ginsburg, Breyer & Sotomayor dissenting).
[26] *Id.* at 3.
[27] *Id.* at 6 (quoting Smith v. Mitchell, 437 F.3d 884 (2006) at 890).

"there was no evidence in the brain itself of the cause of death" was "simply false" and that there "*was* 'evidence in the brain itself'" (emphasis in original).[28] In support of this claim, the majority cited evidence of subdural, subarachnoid, optic nerve and interhemispheric bleeding. However, these findings are *outside* the brain and are associated with a multitude of nontraumatic causes. The majority went on to say that these affirmative indications of trauma formed the basis of the experts' opinions that the child died from shaking so severe "that his brainstem tore."[29] The autopsy did not, however, find any tears in the brainstem, which was not examined microscopically since the pathologists felt that they "wouldn't have seen anything anyway."[30] In short, the Supreme Court was willing to send Ms. Smith – a grandmother described as "warm hearted, sensitive, and gentle" – back to prison to serve a sentence of 15 years to life based on an injury that no one could find.[31] Ultimately, the majority suggestion that clemency might be appropriate given doubts about guilt prevailed, and Governor Brown granted clemency on April 6, 2012.[32]

To understand how we got to the point where invisible injuries are acceptable as proof beyond a reasonable doubt of murder, one must understand the history of SBS/AHT.

---

[28] *Id.* at 7.

[29] *Id.* at 7.

[30] *Id.* at 1 (dissent).

[31] *Id.* at 4. This case was not so much an endorsement of the SBS hypothesis as an expression of the deference the law gives to evidence accepted by a jury, including medical opinions – even speculative and unproven ones – in criminal cases. The majority emphasized that it was bound by legal principles requiring deference to jury verdicts, especially in federal habeas corpus review of state court convictions. To the extent the Court commented on the science, it suggested that there was indeed considerable reason to doubt the medical opinions and conviction. The dissent pointed out expressly that changes in the medical literature since the child's death in 1996 cast considerable doubt on the conviction and the SBS theories underlying it. *Id.* at 4-6. Even the majority acknowledged that "[d]oubts about whether Smith is in fact guilty are understandable," and lamented that "the inevitable consequence of this settled law [of deference to juries] is that judges will sometimes encounter convictions that they believe to be mistaken, but that they must nonetheless uphold." *Id.* at 1, 7.

[32] Carol J. Williams, *Brown commutes sentence of woman convicted of killing grandson*, L. A. TIMES, Apr. 7, 2012 at http://www.latimes.com/news/local/la-me-shaken-baby-clemency-20120407,0,3766620.story. In a review of the medical evidence prior to the grant of clemency, a pathologist at the Los Angeles County coroner's office described eight "diagnostic problems" with the coroner's original ruling that the child had died from violent shaking or a blow to the head. He wrote that the "conservative approach would be to acknowledge these unknowns. The cause of death should be diagnosed as undetermined." Joseph Shapiro & A.C. Thompson, *New Evidence in High-Profile Shaken Baby Case*, NATIONAL PUBLIC RADIO, Mar. 29, 2012 at http://www.npr.org/2012/03/29/149576627/new-evidence-in-high-profile-shaken-baby-case.

B. A Brief History of SBS/AHT

1. ***The Origins.*** For time immemorial, seemingly healthy infants have collapsed or died without any known medical explanation.[33] In the early 1970s, Dr. Guthkelch (a British neurosurgeon) and Dr. Caffey (an American pediatric radiologist) suggested that shaking might explain the unexpected collapse or death of a subset of infants who presented with subdural hemorrhage but typically had no external signs of injury. While shaking was at that time viewed as benign – in one of Dr. Guthkelch's examples, the parent was attempting to save a child from choking – Dr. Guthkelch was concerned that the whiplash effect of shaking could produce subdural hematomas in infants, especially given their weak neck muscles and relatively large heads.[34] In 1974, Dr. Caffey described a two-part sequence in which shaking causes an infant's head to strike its chest and back in "rapid, repeated, to-and-fro, alternating, acceleration-deceleration flexions."[35] Like Dr. Guthkelch, Dr. Caffey was concerned that parents and caretakers did not realize the dangers of shaking, and he recommended a nationwide education campaign to warn of the potential consequences of any action in which the heads of infants were jerked and jolted.[36]

Over the years, the shaking/whiplash hypothesis evolved into the medicolegal hypothesis of "shaken baby syndrome." This hypothesis held that shaking may cause a "triad" of medical findings – subdural hemorrhage, retinal hemorrhage and encephalopathy (brain damage) – and that in the absence of other known explanations, it may be safely inferred from these findings that the child has been shaken.[37] While this conclusion was sometimes supported by other signs of physical injury, such as bruises or fractures, there were often no signs of trauma. In other cases, only one or two elements of the triad were present.[38]

---

[33] *See, e.g.,* D. L. Russell-Jones, *Sudden Infant Death in History and Literature*, 60 ARCHIVES OF DISEASE IN CHILDHOOD 278 (1985).

[34] A. N. Guthkelch, *Infantile Subdural Haematoma and its Relationship to Whiplash Injuries*, 2 BR. MED. J. 430 (1971). As Dr. Guthkelch recently told NPR, at that time in Northern England, parents sometimes punished their children by shaking them, which was considered socially acceptable. Joseph Shapiro, *Rethinking Shaken Baby Syndrome*, NATIONAL PUBLIC RADIO , June 29, 2011 *at* http://www.npr.org/2011/06/29/137471992/rethinking-shaken-baby-syndrome.

[35] John Caffey, *The Whiplash Shaken Infant Syndrome: Manual Shaking by the Extremities with Whiplash-Induced Intracranial and Intraocular Bleedings, Linked with Residual Permanent Brain Damage and Mental Retardation*, 54 PEDIATRICS 396, 401 (1974).

[36] *Id.* at 402-403.

[37] *See, e.g.,* Brian Holmgren, *Prosecuting the Shaken Infant Case, in* THE SHAKEN BABY SYNDROME: A MULTIDISCIPLINARY APPROACH 275, 306 (Stephen Lazoritz & Vincent J. Palusci eds., 2001) ("retinal hemorrhages, bilateral subdural hematoma, and diffuse axonal injury are highly specific for SBS as a mechanism").

[38] *See, e.g.*, Cavazos v. Smith, *supra* note 20 (affirming conviction in SBS case involving "minimal subdural and subarachnoid hemorrhages" but no retinal hemorrhages or brain swelling); Hess v. Tilton, No. CIV S-07-0909, 2009 WL 577661

In the absence of other signs of trauma, the SBS diagnosis was based on the belief that the triad elements were in and of themselves traumatic in origin. Specifically, subdural hemorrhages were attributed to the traumatic rupture of the bridging veins that convey blood from the brain to the large veins (or sinuses) in the fibrous dura lining the skull. Retinal hemorrhages were similarly attributed to the traumatic rupture of retinal blood vessels, while encephalopathy (brain damage) was attributed to the traumatic rupture of the axons (nerve fibers) that connect the nerve cells throughout the brain. Because the brain damage was often bilateral and widespread, it was assumed that the force needed to cause these findings was comparable to or greater than that found in multistory falls or motor vehicle accidents.[39] Thus, if the history provided by the caretakers did not include a major accident, the history was considered to be inconsistent with the findings, and abuse was considered to be the only plausible explanation.[40] In children who had no external signs of trauma, it was further hypothesized that the abuse must have consisted of violent shaking.

A corollary of the SBS hypothesis – and one that was particularly important for the legal system – was that the injury could be timed and the perpetrator identified based solely on the medical findings. Since the

---

(E.D. Cal., Mar. 5, 2009) (affirming conviction in SBS case involving brain swelling and retinal hemorrhages but no subdural hemorrhage).

[39] *See* Mary E. Case et al., *Position Paper on Fatal Abusive Head Injuries in Infants and Young Children,* 22 AM. J. FORENSIC MED. PATHOL. 112, 120 (2001) ("fatal accidental shearing or diffuse brain injuries require such extremes of rotational force that they occur only in obvious incidents such as motor vehicle accidents. Besides vehicular accidents, other fatal accidental childhood head injuries tend to involve crushing or penetrating trauma, which is readily evident. These injuries tend to be the result of falling from considerable heights (greater than 10 feet) or having some object penetrate the head"); Alex Levin et al., Clinical Statement, *Abusive Head Trauma/ Shaken Baby Syndrome*, AM. ACADEMY OPHTHALMOLOGY, available at http://one.aao.org/ce/practiceguidelines/clinicalstatements_content.aspx?cid=914163d5-5313-4c23-80f1-07167ee62579 (retinal hemorrhages typical of AHT/SBS are uncommon in severe accidental head trauma such as falls from a second-story level or a motor vehicle collision).

[40] *See, e.g.*, Edward J. Imwinkelried, *Shaken Baby Syndrome: A Genuine Battle of the Scientific (and Non-Scientific) Experts,* 46 CRIM. L. BULL. 6 (2010) and cases cited therein (noting that "the most common analogies [used by prosecution experts] are to the amount generated by high speed automobile accidents and a fall from a several-story building. The experts analogize to these "real-life accident scenarios" in order to give the trier of fact a sense of the 'massive, violent' force required to produce this kind of brain injury"); cited cases include Mitchell v. State, 2008 WL 316166 (Ark. Ct. App. 2008) (examining pediatrician equated the force necessary to produce the triad with that of a high-speed automobile accident); People v. Dunaway, 88 P.3d 619, 631, 632 (Colo. 2004) (prosecution expert stated that subdural hemorrhages occur in "such things as falling from a several story building or being in a high speed motorcycle accident or a child say is on a bicycle hit by a car...when we see subdurals in accidental injury, it's from a major trauma. It requires massive force"); In re Child, 25 Misc. 3d 745, 2008 WL 6572459 (N.Y. Fam. Ct. 2008) (prosecution expert stated that SBS findings "simulate being in a car crash at 'around 35 to 40 miles per hour'").

damage caused by the traumatic rupture of nerve fibers throughout the brain would be devastating with immediate loss of function (as in concussion), there could be no period of relative normality ("lucid interval") following the injury. It was therefore widely accepted that the last person with the baby must have been responsible.[41] In effect, SBS quickly became a criminal category of *res ipsa loquitur* cases, *i.e.*, cases in which "the thing speaks for itself." This eliminated the need for any additional evidence, including motive or history of abuse, and resulted in quick, easy and virtually routine convictions of parents and caretakers based solely on the medical testimony of prosecution experts.[42]

Given the underlying assumptions of the SBS hypothesis, the suggestion that birth injuries, short falls, or natural causes could result in the triad, or that a child might have a lucid interval after such an injury, was viewed as heretical: how could birth injuries produce findings that did not become apparent for days, weeks or months after birth? How could short falls produce traumatic findings akin to – or worse than – those seen in major motor vehicle accidents and multistory falls? How could a natural disease process rupture veins and axons, causing diffuse traumatic brain injury? And how could there be a lucid interval after bridging veins had been ruptured and axons torn throughout the brain? Not surprisingly, those who suggested such possibilities were often disparaged or vilified.[43] Unfortunately, those attacks continue to this day.[44]

---

[41] *See, e.g.*, Deborah Tuerkheimer, *The Next Innocence Project: Shaken Baby Syndrome and the Criminal Courts*, 87 WASH. UNIV. L. REV. 1, 18 (2009) (noting that parents and caretakers have been accused of shaking the child in their care because they were present immediately before the child's loss of consciousness).

[42] *See, e.g.*, Imwinkelried, *supra* note 36 ("it seems clear that during the past two decades, prosecution expert testimony about shaken baby syndrome has contributed to thousands of convictions").

[43] Those who question the scientific basis for SBS/AHT are routinely accused of incompetence, greed, indifference to child abuse and, more recently, of possibly having histrionic/borderline personality disorders. *See, e.g.*, Christopher Spencer Greeley, Presentation, *Dissent or Denialism?: A Scholarly Misadventure with the Medical Literature (and the Media)*, New York City Abusive Head Trauma/Shaken Baby Syndrome Training Conference (Sept. 23, 2011) at http://www.queensda.org/SBS_Conference/Denialism&TheMedicalLiterature,0911,NYC,Handout.pdf (suggesting that researchers who question SBS/AHT theory use "sleaze tactics" and may have "histrionic/borderline" personality disorders); Brian Holmgren, Presentation, *To Tell the Truth – Examining Defense Witness Testimony in Abusive Head Trauma Cases*, Eleventh International Conference on Shaken Baby Syndrome/Abusive Head Trauma, sponsored by the National Center on Shaken Baby Syndrome (Sept. 2010) (showing excerpts of testimony from defense experts juxtaposed with an image of Pinocchio with a growing nose at a keynote presentation teaching doctors and prosecutors how to discredit defense witnesses; this presentation concluded with a sing-along to the tune of "If I only had a brain" led by a prominent child abuse pediatrician, joined by prosecutors and doctors, mocking those who propose diagnostic alternatives to SBS/AHT) brochure at http://www.dontshake.org/pdf/Program_Atlanta2010_8-18-10%20v2.pdf (presentation notes and lyrics on file with authors); Robert M. Reece et al., *Response to Editorial*

2. ***The warnings.*** Despite its popularity, there were early warning signs that the SBS hypothesis might be flawed. The first serious warning arose in 1987, when Dr. Duhaime, a young neurosurgeon, and several biomechanical engineers attempted to validate the SBS hypothesis by measuring the force of shaking and comparing it to accepted head injury thresholds. While crude, these early experiments indicated that the force generated by shaking an infant was well below established head injury criteria and was only approximately one-fiftieth the force generated by impact.[45] This study concluded that,

> [T]he shaken baby syndrome, at least in its most severe acute form, is not usually caused by shaking alone. Although shaking may, in fact, be part of the process, it is more likely that such infants suffer blunt impact . . . . Unless a child has predisposing factors such as subdural hygromas, brain atrophy, or collagen-vascular disease, fatal cases of the shaken baby syndrome are not likely to occur from the shaking that occurs during play, feeding, or in a swing, or even from the more vigorous shaking given by a caretaker as a means of discipline.[46]

Dr. Duhaime later suggested that the triad was likely caused by shaking followed by impact, possibly on a soft padded surface.[47]

Further warnings arose during the 1997 Louise Woodward trial, popularly known as the "Boston nanny case."[48] In *Woodward*, Dr.

---

from 106 Doctors, 328 BRIT. MED. J. 1316, 1316 (2004) (arguing that SBS skeptics have a "worrisome and persistent bias against the diagnosis of child abuse in general"). Personal and professional attacks of this nature have made scientific debate difficult.

[44] While Dr. Narang does not endorse these attacks, he does suggest, without offering evidence, that those who point out flaws in the SBS diagnosis or identify alternative causes are motivated by monetary gain. Narang, *supra* note 3, at 592 ("[T]he pecuniary interest in providing expert testimony cannot be underestimated. It has posed and continues to pose a significant risk to the presentation of unbiased medical information"). In our experience, the marginal income for defense experts is generally small relative to the workload and the hostility encountered in the courtroom and professional settings. Because the funding is often inadequate, defense experts often provide pro bono reports and/or testimony based on the research in their own specialties.

[45] Ann-Christine Duhaime et al., *The Shaken Baby Syndrome a Clinical Pathological and Biomechanical Study*, 66 J. NEUROSURG. 409 (1987).

[46] *Id.* at 414.

[47] *See, e.g.*, A. C. Duhaime et al., *Head Injury in Very Young Children: Mechanisms, Injury Types, and Ophthalmologic Findings in 100 Hospitalized Patients Younger Than 2 Years of Age*, 90 PEDIATRICS 179, 183 (1992) (in "shaken impact syndrome," head injury is caused by rapid angular deceleration to the brain through impact after a shaking episode; if the head strikes a soft padded surface, contact forces will be dissipated over a broad area and external or focal injuries may be undetectable).

[48] *See* Commonwealth v. Woodward, 694 N.E.2d 1277 (1998); *See, e.g.*, Carey Goldberg, *Massachusetts High Court Backs Freeing Au Pair in Baby's Death*, N. Y.

Patrick Barnes, a pediatric neuroradiologist then at Harvard and one of the co-authors of this article, testified for the prosecution.[49] In the same case, several credible and well-established experts presented, perhaps for the first time, serious alternatives to the SBS hypothesis. At the trial, Dr. Jan Leestma, the author of *Forensic Neuropathology*, Dr. Michael Baden, a well-known forensic pathologist, and Dr. Ronald Uscinski, a Georgetown neurosurgeon, testified that the child had a chronic (old) subdural hemorrhage that rebled.[50] At the time, this was viewed as a "courtroom diagnosis" and its proponents were attacked by supporters of the SBS hypothesis.[51] Today, however, rebleeding from a chronic subdural hemorrhage is widely accepted, even by supporters of the SBS/AHT hypothesis.[52] Following the *Woodward* case, a number of forensic pathologists questioned the validity of the SBS diagnosis, with one leading forensic pathologist urging his colleagues to refrain from the type of "dramatic, unscientific" remarks that were permeating courtroom testimony, such as the standard phrase "the equivalent of a fall from a two-story building."[53]

3. *2001: a developing schism.* The public airing of the issues in the *Woodward* case led to a renewed interest in SBS among researchers. In 2001, Dr. Geddes, a British neuropathologist, and her colleagues published careful studies of the brains of infants who had reportedly died from abuse.[54] The results of these studies were unexpected.[55] In the first study ("Geddes I"),[56] the researchers found

---

TIMES (June 17, 1998) *at* http://www.nytimes.com/1998/06/17/us/massachusetts-high-court-backs-freeing-au-pair-in-baby-s-death.html?ref=louisewoodward.

[49] Like many others, Dr. Barnes has revisited these issues since 1997, with particular emphasis on the teachings of evidence based medicine and the correlation between the neuroradiology and neuropathology of the infant brain.

[50] The Woodward case also involved a skull fracture, making timing difficult. *See* Special Report, *Timetable of Woodward Case,* BBC NEWS (Nov. 10, 1998) *at* http://news.bbc.co.uk/2/hi/special_report/louise_woodward_case/29232.stm.

[51] David L. Chadwick et al., *Shaken Baby Syndrome—A Forensic Pediatric Response*, 101 PEDIATRICS 321 (1998).

[52] *See, e.g.,* Marguerite M Caré, *Neuroradiology, in* ABUSIVE HEAD TRAUMA IN INFANTS AND CHILDREN, A MEDICAL, LEGAL, AND FORENSIC REFERENCE 73, 81 (2006) (septations or membranes that develop within chronic hematomas may predispose infants to repeated episodes of bleeding within these collections; such rebleeding can occur with little or no trauma).

[53] Cyril H. Wecht, *Shaken Baby Syndrome, Letter to the Editor,* 20 AM. J. FORENSIC MED. PATHOL. 301 (1999); *see also* John Plunkett, *Shaken Baby Syndrome and the Death of Matthew Eappen a Forensic Pathologist's Response,* 20 AM. J. FORENSIC MED. PATHOL. 17 (1999). As discussed below, forensic pathologists have always been more skeptical of the SBS hypothesis than other specialties, particularly pediatricians.

[54] David I. Graham, *Editorial: Paediatric Head Injury*, 124 BRAIN 1261, 1261 (2001) (Geddes and her colleagues conducted a "meticulous clinicopathological correlation in 53 cases of non-accidental paedatric head injury").

[55] Dr. Geddes has described her surprise that the microscopic examinations failed to find the widespread and severe traumatic brain damage assumed to be present in shaken infants. Jennian Geddes, *Questioning Traditional Assumptions*, BARTS AND THE

that the brain pathology was predominantly hypoxic or ischemic (*i.e.*, due to lack of an oxygenated blood supply) rather than traumatic in nature. Unlike the traumatic hemorrhages found in adults and older children, moreover, the subdural hemorrhages in allegedly abused infants were typically thin and trivial in quantity – containing far less blood than would be expected from ruptured bridging veins, as hypothesized in SBS. While some infants showed evidence of localized axonal injury to the craniocervical junction or cervical cord, the majority did not, casting further doubt on the SBS hypothesis. In the second study ("Geddes II"), Dr. Geddes and her colleagues described the scientific evidence supporting a traumatic origin for the brain damage in allegedly abused children as "scanty." In many respects, the findings in these children were virtually indistinguishable from the findings in infants who had died natural deaths.[57]

While far from dispositive, the implications of Geddes I and II were devastating: if Dr. Geddes and her colleagues were correct, the SBS hypothesis, which rested on the notion that the triad was caused by the traumatic tearing of veins and axons, was likely wrong. While traumatically torn axons are by definition caused by trauma, there are many non-traumatic causes for hypoxic axonal injury. The brain may, for example, be deprived of oxygen because the heart or lungs are not functioning properly or because the child is suffering from widespread infection (sepsis). This research raised, for the first time, the possibility that the brain findings that had been attributed to traumatically torn axons from violent shaking might reflect hypoxia-ischemia from any medical condition that affected the flow of oxygen to the brain. Dr. Geddes' research also raised problems with timing: if the brain damage was secondary to the deprivation of oxygenated blood from any source, the ensuing brain swelling could develop quickly or slowly, over a period of hours to days, with collapse occurring whenever the brain's basic needs were no longer met by the dwindling supply of oxygenated blood. Although Geddes I and II were heavily criticized at the time, it is now widely accepted that the brain swelling seen in allegedly shaken infants is hypoxic-ischemic rather than traumatic in nature.[58]

LONDON CHRONICLE, Spring 2006, *at* http://www.qmul.ac.uk/alumni/publications/blc/blc_spring06.pdf.

[56] J. F. Geddes et al., *Neuropathology of Inflicted Head Injury in Children, I. Patterns of Brain Damage,* 124 BRAIN 1290 (2001).

[57] J.F. Geddes et al., *Neuropathology of Inflicted Head Injury in Children, II. Microscopic Brain Injury in Infants,* 124 BRAIN 1299, 1299, 1304 (2001).

[58] *See, e.g.,* Mark S. Dias, *The Case for Shaking, in* CHILD ABUSE AND NEGLECT, DIAGNOSIS, TREATMENT AND EVIDENCE 364, 370 (Carole Jenny, ed., 2011) (it is increasingly clear from neuroimaging studies and post-mortem analyses that the widespread cerebral and axonal damage in AHT cases is ischemic rather than directly traumatic); Neil Stoodley, *Non-accidental head injury in children: gathering the evidence,* 360 THE LANCET 272 (2002) (noting the growing evidence that hypoxic-ischaemic damage is of greater importance than traumatic axonal or shearing injury in the pathophysiology of nonaccidental head injury).

Biomechanical objections to the SBS hypothesis also returned to the forefront in 2001. In April, Professor Werner Goldsmith, a professor of biomechanical engineering at the University of California at Berkeley, raised the biomechanical concerns with the National Institutes of Health (NIH). In his presentation, Professor Goldsmith noted that while the vast majority of pediatric head injuries were accidental, others resulted from abuse or physiological (natural) causes, unaccompanied by mechanical trauma.[59] Given the difficulty of determining causation, he urged the development of more sophisticated biomechanical models and more reliable head injury criteria for infants. He also urged biological specialists, medical professionals and biomechanicians to collaborate in investigating the properties of the immature infant brain and surrounding blood vessels that might make them more susceptible to trauma.[60] Such a program, Professor Goldsmith suggested, would "enormously reduce the number of cases now brought into criminal courts, and the concomitant costs, estimated to be in the multiple millions of dollars, as well as avoid the true trauma, emotionally, financially, and temporally, of individuals falsely accused of abuse when the occurrence was accidental."[61]

In the same year, Dr. John Plunkett, a forensic pathologist, published an article on fatal short falls from playground equipment.[62] While most of the children were older than typical SBS infants, his report included a videotaped fall of a toddler from a plastic indoor play gym that resulted in the triad findings and death after a short lucid interval. This videotape provided seemingly indisputable proof that the triad could result from falls of less than three feet and that lucid intervals could occur.[63]

---

[59] Werner Goldsmith, Presentation, *Biomechanics of Traumatic Brain Injury in Infants and Children*, NAT. INSTITUTES OF HEALTH (April 2001) (on file with authors). As Professor Goldsmith recognized, "head injury" includes any insult to the brain, whether from accidental, abusive or natural causes. This terminology often causes confusion in the literature.

[60] Professor Goldsmith specifically urged research on the rate of blood absorption and effusion of ruptured blood vessels, which is the subject of the Squier & Mack papers (discussed below).

[61] *Id.*

[62] John Plunkett, *Fatal Pediatric Head Injuries Caused by Short-Distance Falls*, 22 AM. J. FORENSIC MED. PATHOL. 1 (2001).

[63] *Id.* at 4. In this case the child's feet were 28" above the floor when she fell; medical records showed a large subdural hemorrhage, bilateral retinal hemorrhages and extensive edema. In the past year, two other videotaped fatal short falls resulting in death have been reported. One was of an infant who fell from a Kroger shopping cart onto concrete in Macon, Georgia, caught on surveillance video (John Stevens, *Three-Month-Old Boy Dies After Falling Out of Shopping Cart as Mother Walked Back to Car*, DAILY MAIL, September 22, 2011, at www.dailymail.co.uk/news/article-2040559). The other was a fall onto a mat at an indoor mall playground shown by the Queens District Attorney's Office at the 2011 New York City Abusive Head Trauma/Shaken Baby Syndrome Training Conference (Sept. 22, 2011).

By this time, however, the SBS hypothesis had taken on a life of its own. By 2001, shaking as the primary or exclusive cause of the triad had been taught in the medical schools for decades, not as a hypothesis but as scientific fact. Prosecutions were well-publicized, and an effective advocacy group was training social workers and prosecutors to identify, prosecute and win cases against parents and caretakers who had allegedly shaken their children.[64] Doctors affiliated with this group also produced SBS position papers for the major medical associations. In 2001, the National Association of Medical Examiners (NAME) – the professional association for forensic pathologists – published an article entitled "Position Paper on Fatal Abusive Head Injuries in Infants and Young Children," which incorporated the SBS hypothesis.[65] Although this paper did not pass peer review and was not endorsed by the membership,[66] it was published in the NAME journal, accompanied by a somewhat ambiguous and little-heeded editorial caveat.[67] In the same year, the Committee on Child Abuse and Neglect of the American Academy of Pediatrics (AAP) published a similar paper, entitled "Shaken Baby Syndrome: Rotational Cranial Injuries—Technical Report."[68] The AAP paper recommended a presumption of child abuse whenever a child younger than 1 year suffers an intracranial injury. While the NAME paper is no longer in effect and the AAP paper has

---

[64] The National Center on Shaken Baby Syndrome (NCSBS) began offering SBS prevention programs in 1990 and incorporated as a legal entity in 2000. According to its website, the NCSBS reaches thousands of medical, legal, child protection and law enforcement professionals every year. *See* NCSBS website at http://dontshake.org/.

[65] Case, *supra* note 35.

[66] E-mail from Dr. DiMaio, Editor of the *American Journal of Forensic Medicine and Pathology*, to Dr. Plunkett (March 6, 2003) ([T]he "position paper: was reviewed by peer reviewers and determined not to be a position paper but an ordinary article expressing the opinion of the authors . . . The paper [does] not meet the criteria of a position paper . . . . Calling a tail a leg does not make it one."); Email from Vincent DiMaio to NAME-L@Listserve.cc.emory.edu (Feb. 7, 2002) ("As editor of the AJFMP, I had serious misgiving about publishing this paper, not because of its contents but in that it is described as a position paper . . . . If one bothers to read the box in the lower left corner of the first page of the article, one will see that the paper was rejected as a position paper by the three reviewers . . . . As an aside, the paper in its original form was rejected by 4 of 5 reviewers . . . . Shaken baby syndrome is controversial in that a number of individuals doubt its existence . . .") (e-mails on file with authors).

[67] Case, *supra* note 35, at 112 ("Editor's note: The Board of Directors of the National Association of Medical Examiners charged the authors of this article with writing a position paper on the shaken baby syndrome. This article was the result. The manuscript was reviewed by three reviewers on the Board of Editors of the American Journal of Forensic Medicine and Pathology. They believed that while it was worthy of publication, it should not be published as a position paper because of the controversial nature of the subject. The Board of Directors responded to this opinion by stating that position papers always deal with controversial subjects").

[68] Comm. on Child Abuse and Neglect, Am. Acad. of Pediatrics, *Shaken Baby Syndrome: Rotational Cranial Injuries – Technical Report,* 108 PEDIATRICS 206 (2001).

been substantially modified,[69] these papers gave an imprimatur of scientific and medical endorsement to the SBS hypothesis that was accepted, largely uncritically, by the medical and legal communities.

4. ***A decade of debate.*** The decade following the Geddes and Plunkett papers and the NAME/AAP position papers was filled with raucous debate, sometimes more rhetorical than substantive. However, a few key points emerged.

a. **2002 NIH conference.** In 2002, NIH held a conference to address the disputed issues.[70] By this time, the terminology was shifting away from shaken baby syndrome to more generalized terms, such as inflicted neurotrauma and abusive head trauma. Although the conference was limited to supporters of the SBS/AHT hypothesis, the lack of evidentiary support for SBS was repeatedly acknowledged, beginning in a preface to the conference proceedings by Dr. Carol Nicholson, a Program Director at NIH:

> The debate over "shaken baby syndrome" continues to rage in our country. Because there is very little scientific experimental or descriptive work, the pathophysiology remains obscure, and the relationship to mechanics even cloudier.... What we need is science—research and evidence that just isn't there right now. The evidence that does exist has not been subjected to evidence-based scrutiny in a multidisciplinary scientific forum.[71]

Dr. Robert Reece, a Clinical Professor of Pediatrics, made similar points in his preface:

> There have been numerous conferences on this subject over the past several years, but to date, none of these has made the analysis of evidence-based literature the mission of the conference. What literature is there that is based on well-designed studies? How many of the more than 600 peer-reviewed articles in the medical literature can withstand the scrutiny of evidence-based analysis?[72]

---

[69] As addressed below, the NAME paper was withdrawn in 2006; the AAP paper was modified in 2009.

[70] *Inflicted Childhood Neurotrauma*, 2003 PROC. OF A CONFERENCE SPONSORED BY DEPARTMENT OF HEALTH AND HUMAN SERVICES, NATIONAL INSTITUTES OF HEALTH, NATIONAL INSTITUTE OF CHILD HEALTH AND HUMAN DEVELOPMENT, OFFICE OF RARE DISEASE AND NATIONAL CENTER FOR MEDICAL REHABILITATION RESEARCH. These conference proceedings are one of the two treatises referenced by Narang, *supra* note 3, at 538-539.

[71] *Id.* at IX (noting that the escalating emotional and forensic advocacy was proving destructive).

[72] *Id.* at VIII.

Dr. Reece emphasized that much of the literature was based on clinical phenomena rather than "bench research" and that the contributions of basic scientists doing research on the physiology and pathophysiology of the central nervous system were essential to understanding these issues.[73] He also made clear that much of what was being considered at the conference was based on "a preponderance of the evidence" rather than "evidence beyond a reasonable doubt" – the standard required in criminal cases.[74]

Other conference participants addressed the new literature. Although SBS theory had previously held that short falls were benign, Dr. Feldman advised that in a few cases short falls "may be fatal or have residual effects."[75] Dr. Sege noted that while some might argue that additional research, which he characterized as a "massive undertaking," would simply confirm the current SBS/AHT understandings, "[s]adly, the history of medicine is littered with things known to be true at the time that weren't."[76] Dr. Christian mounted a spirited defense of SBS/AHT theory, claiming that "[h]omicide is the leading cause of injury death in infancy," but agreed with Dr. Sege that "[t]he literature is replete with case reports of medical diseases that have been misdiagnosed as child abuse."[77]

The conference participants generally agreed that, despite its volume, the SBS/AHT literature suffered from serious gaps. Dr. Hymel noted that the peer-reviewed SBS/AHT medical literature "largely represents Class 3 scientific evidence from retrospective case series" and "contains little if any firsthand clinical information from admitted perpetrators of inflicted childhood neurotrauma, and no data regarding the *reliability* and/or *validity* of the acute clinical information provided by admitted perpetrators of inflicted neurotrauma."[78] Dr. Duhaime warned that SBS/AHT presented a complex puzzle that had been incompletely modeled and that a great deal of work needed to be done using tissues, animals, mathematical models and human observations, superimposed on age-dependent changes and physiological thresholds.[79]

---

[73] *Id.*
[74] *Id.*
[75] *Id.* at 33.
[76] *Id.* at 40-41.
[77] *Id.* at 43.
[78] *Id.* at 67. As discussed below, under the standards of evidence-based medicine, the available evidence is ranked in four categories, starting with randomized controlled trials (Class 1), which are the most comprehensive and the most reliable, and ending with case studies (Class 4), which may provide valuable but limited insights. Class 3 evidence includes case-control studies and non-consecutive studies with inconsistently applied reference standards. *See* Bob Phillips, Chris Ball, Dave Sackett, Doug Badenoch, Sharon Straus, Brian Haynes and Martin Dawes, *Levels of Evidence*, OXFORD CENTRE FOR EVIDENCE-BASED MEDICINE (Updated by Jeremy Howick, Mar. 2009) at http://www.cebm.net/index.aspx?o=1025.
[79] *Id.* at 253.

Dr. Jenny identified the methodological difficulties with the existing literature:

> One resounding criticism in this body of literature poses a methodological dilemma when attempting to study mode of presentation of inflicted head trauma. This dilemma is the problem of circularity of reasoning. That is, we use certain predetermined, generally accepted criteria to determine if a child's injuries are inflicted or unintentional, such as delay in seeking care and presence of retinal hemorrhages. Then, when we describe the mode of presentation, those criteria are found to occur most frequently in abused children. A most sticky methodological question is, "What is the gold standard in determining if a child is abused prior to assigning that child to a study cell?" Careful definitions of standards for determining abuse are needed.[80]

Dr. Dias, a conference organizer, agreed that there was "some degree of a circularity in reasoning: if one defines a particular injury or pattern of injuries a priori as inflicted, then by definition one will rarely, if ever, ascribe these injuries to…an unintentional mechanism."[81]

b. Biomechanics. In general, the biomechanical literature continued to conclude that shaking was an unlikely cause of the triad. For example, a 2002 biomechanical review concluded that a three-foot fall produces forces approximately ten times greater than shaking; that spontaneous rebleeds may explain the onset of symptoms in children with chronic subdural hemorrhage; that severe shaking would be expected to damage the cervical cord and spine before producing intracranial injuries; and that the levels of force required for shaking to produce retinal bleeding and damage to the eye are biomechanically improbable.[82] These findings were similar to those in a joint study conducted by Dr. Jenny, a leading SBS proponent, and Aprica, a Japanese baby products company that had created a more biofidelic model of the human infant.[83] Other research was in accord: while

---

[80] *Id.* at 51-52. Dr. Jenny identified the studies of Duhaime (1987); Ewing-Cobbs (1998); Reece (2000); and Feldman (2001) as "methodologically superior." *Id.* at 51. Three of these are discussed below.

[81] *Id.* at 100.

[82] A.K. Ommaya et al., *Biomechanics and Neuropathology of Adult and Paediatric Head Injury,* 16 BR. J. NEUROSURG. 220 (2002).

[83] These studies confirmed that the maximum linear acceleration produced by shaking was less than 1/3 that produced by rolling off a sofa and less than 1/10 that of a fall from chest level when being held by an adult. Violent shaking and slamming on a thin carpet over a wood floor was comparable to the chest level fall, while slamming onto a mat without shaking produced a force approximately 50% greater than the fall from chest level. C. Jenny et al., *Development of a Biofidelic 2.5 kg Infant Dummy and Its Application to Assessing Infant Head Trauma During Violent Shaking,* 2002 INJURY

impact reaches known injury thresholds, shaking does not produce the force required to rupture bridging veins and axons and would cause extensive cervical spine injury or failure (*i.e.*, neck injury) before causing such effects.[84] By then, after thirty years, there were still no witnessed accounts of the shaking of a previously well child resulting in the triad, casting further doubt on the mechanism.[85]

c. SBS and evidence-based medicine. The weaknesses in the literature were not passing unnoticed in the outside world. In a 2003 article published in the NAME journal, Dr. Donohoe, a general practitioner in Australia, examined the research support for SBS through 1998 and concluded what others – including the NIH conference participants – had been saying privately for years: the research basis for shaken baby syndrome was remarkably weak.[86] Dr. Donohoe described the evidence for SBS as "analogous to an inverted pyramid, with a small database (most of it poor-quality original research, retrospective in nature, and without appropriate control groups) spreading to a broad body of somewhat divergent opinions. One may need reminding that repeated opinions based on poor-quality data cannot improve the quality of evidence."[87] He concluded that "the commonly held opinion that the finding of SDH [subdural hemorrhage] and RH [retinal hemorrhage] in an infant was strong evidence of SBS was unsustainable, at least from the medical literature."[88]

d. Alternative diagnoses. Given the biomechanical findings, impact took on new significance as the most likely cause of the triad. But this raised new issues. First, if the triad was caused by impact, why did so few children have external signs of impact, such as fractures or bruises? Second, how much force is required to cause injury from impact? And third, can we reliably distinguish between accidental

---

BIOMECHANICS RESEARCH, PROCEEDINGS OF THE THIRTIETH INTERNATIONAL WORKSHOP, sponsored by the National Highway Traffic Safety Administration (on file with authors).

[84] *See, e.g.*, Michael T. Prange et al., *Anthropomorphic Simulations of Falls, Shakes, and Inflicted Impacts in Infants*, 99 J. NEUROSURG. 143 (2003); Ommaya, *supra* note 78; *see also* R. Uscinski, *Shaken Baby Syndrome: Fundamental Questions* 16 BRIT. J. NEUROSURG. 217, 218 (2002) (biomechanical research has raised questions about whether shaking is the true cause of intracranial injuries in alleged SBS cases); Ronald H. Uscinski, *Shaken Baby Syndrome: An Odyssey*, 46 NEUROLOGIA MEDICO-CHIRURGICA 57, 59 (2006) (SBS-type accelerations should damage the cervical spinal cord and brainstem before head injury is observed).

[85] There are also no reported cases of video recordings capturing violent shaking resulting in the triad. While several caregivers have been caught on videotape shaking infants in their care, to our knowledge none of these children exhibited any of the triad findings, or any injury at all.

[86] Mark Donohoe, *Evidence-Based Medicine and Shaken Baby Syndrome Part I: Literature Review, 1966-1998*, 24 AM. J. FORENSIC MED. PATHOL. 239 (2003). Dr. Narang criticizes Dr. Donohoe's review article and his review of the SBS literature. As discussed *infra*, that criticism mistakes the nature of Dr. Donohoe's inquiry.

[87] *Id.* at 241.

[88] *Id.*

and inflicted impact – and if so, how? Some clinicians and pathologists avoided these issues by redefining the "triad" – which had previously been viewed as diagnostic of shaking – as evidence of impact. At the same time, clinicians quite rightly began to look closely for other possible signs of impact or abuse, ranging from small bruises or discolorations to fractures or other bony abnormalities that might help determine causation.

While some researchers and clinicians struggled to differentiate between accidental and inflicted impact, others began to consider – or more precisely re-consider – the role of natural conditions or birth trauma as causal or contributing factors for the triad. As Dr. Guthkelch noted in 1953, subdural hemorrhages are often associated with abnormal or difficult labor and, less commonly, with venous thrombosis, a form of childhood stroke often associated with infection and/or dehydration.[89] Metabolic disorders, nutritional deficits and infection have also long been recognized as causes of subdural hemorrhage.[90]

During this period, the child abuse literature increasingly recognized alternative causes for subdural hemorrhages and other elements of the triad. In 2002, Drs. Jenny, Hymel and Block – all prominent child abuse pediatricians – published an article identifying a wide range of nontraumatic etiologies for subdural hemorrhages and describing minor accidental injuries confirmed by medical personnel that resulted in intracranial hemorrhage.[91] The article further recognized that older subdural collections can rebleed spontaneously or from minor impact, and that no prospective, comparative studies had measured the frequency or consequences of rebleeding in young children with chronic subdural collections.[92]

In 2003, Dr. Geddes suggested that the subdural and retinal hemorrhages seen in natural deaths and alleged SBS cases may reflect a cascade of events, including raised intracranial pressure, central venous and systemic arterial hypertension, immaturity and hypoxia-related vascular fragility – a suggestion that became known as the "Unified Hypothesis" or Geddes III.[93]

---

[89] A. N. Guthkelch, *Subdural Effusions in Infancy: 24 Cases,* 1 BR. MED. J. 233, 233 (1953) (abnormal or difficult labor present in 75% of the cases in this series).

[90] Narang, *supra* note 3, at 525, footnote 138.

[91] Kent P. Hymel, Carole Jenny & Robert W. Block, *Intracranial Hemorrhage and Rebleeding in Suspected Victims of Abusive Head Trauma: Addressing the Forensic Controversies,* 7 CHILD MALTREATMENT 329, 331, 332, 342-343 (2002) (causes for subdural hemorrhage include prenatal, perinatal and pregnancy-related conditions; birth trauma; metabolic or genetic disease; congenital malformations; oncologic disease; autoimmune disorders; clotting disorders; infectious disease; poisons, toxins or drugs; and other miscellaneous conditions).

[92] *Id* at 342, 344.

[93] J. F. Geddes et al., *Dural Haemorrhage in Non-Traumatic Infant Deaths: Does It Explain the Bleeding in 'Shaken Baby Syndrome'?,* 29 NEUROPATHOL. APPL. NEUROBIO. 14, 19 (2003).

By 2006, it was widely recognized by supporters of the SBS/AHT hypothesis that there are many "mimics" of SBS/AHT, including accidental causes and a variety of illnesses and medical conditions, ranging from birth trauma to childhood stroke.[94] Since then, other studies have continued to add to our knowledge. For example, a study by Dr. Rooks and her colleagues found that approximately 46% of asymptomatic newborns had thin subdural hemorrhages, confirming that subdural hemorrhages are not necessarily symptomatic and do not necessarily (or even generally) cause long lasting problems.[95] Another study found a clear correlation between intradural/subdural hemorrhage and the degree of hypoxia in neonates.[96] Today, every month seems to bring forth new articles and commentary, adding to the available information but also increasing the confusion. Like Dr. Narang, we do not attempt to review all of these studies but rather address key new articles by subject, noting only that the list of possible causes for findings previously viewed as diagnostic of abuse continues to expand.

e. The position papers revisited. By 2006, it was evident that the literature on pediatric head injury no longer supported the assumptions underlying the SBS hypothesis and that the major medical associations would have to revise their position papers. This process has resulted in considerable confusion within the medical profession and very little guidance on the proper approach to diagnosis.

In October 2006, the NAME Board of Directors withdrew its "Position Paper on Fatal Abusive Head Injuries in Infants and Young Children."[97] Although no explanation was offered, the NAME conference of the same date included presentations entitled "Use of the Triad of Scant Subdural Hemorrhage, Brain Swelling, and Retinal

---

[94] By 2006, the alternative causes or "mimics" included prenatal and perinatal conditions; congenital malformations; genetic conditions; metabolic disorders; coagulation disorders, including venous sinus thrombosis (a form of childhood stroke); infectious disease; vasculitis; autoimmune conditions; oncology; toxins and poisons; nutritional deficiencies; and complications from medical-surgical procedures. *See* Sirotnak, *supra* note 10; Dowd, *supra* note 10.

[95] V. J. Rooks et al., *Prevalence and Evolution of Intracranial Hemorrhage in Asymptomatic Term Infants*, 29 AM. J. NEURORADIOL. 1082, 1085 (2008). While most of these subdural hemorrhages disappeared within the first month, one had evidence of new subdural bleeding at two weeks, with subdural fluid collections still evident at four weeks. With a larger study population, more variations might be expected.

[96] Marta C. Cohen & Irene Scheimberg, *Evidence of Occurrence of Intradural and Subdural Hemorrhage in the Perinatal and Neonatal Period in the Context of Hypoxic Ischemic Encephalopathy: An Observational Study from Two Referral Institutions in the United Kingdom*, 12 PEDIATRIC & DEVELOPMENTAL PATHOLOGY 169 (2009) (finding a clear correlation between intradural/subdural hemorrhage and the degree of hypoxia in neonates, with bleeding in the parietal dura developing with more severe or prolonged hypoxia).

[97] E-mail from Gregory G. Davis, Bd. of Directors, NAME, to John Plunkett, MD, and R. Wright (Oct. 17, 2006) (on file with authors). The 2001 NAME position paper had originally been scheduled to sunset in 2006; however, the Board had extended it to 2008. In October 2006, the Board rescinded the renewal.

Hemorrhages to Diagnose Non-Accidental Injury is Not Scientifically Valid" and "'Where's the Shaking?' Dragons, Elves, the Shaking Baby Syndrome and Other Mythical Entities."[98] No subsequent NAME paper has been approved, leaving it to individual forensic pathologists to reach their own interpretations on causality without guidance from their association. Not surprisingly, this has produced inconsistent conclusions. Today, based on similar or even identical medical findings, some forensic pathologists still endorse shaking as the causal mechanism, others diagnose blunt force trauma (*i.e.*, impact, accidental or abusive) and yet others consider a wide range of possibilities, including natural causes. In Professor Tuerkheimer's words, such variances produce "fluky justice."[99]

In 2009, the AAP replaced its technical report on Shaken Baby Syndrome with a policy statement entitled "Abusive Head Trauma in Infants and Children."[100] The authors stated that though the term shaken baby syndrome is often used by physicians and the public, "advances in the understanding of the mechanisms and clinical spectrum of injury associated with abusive head trauma compel us to modify our terminology to keep pace with our understanding of pathological mechanisms. Although shaking an infant has the potential to cause neurologic injury, blunt impact or a combination of shaking and blunt impact can cause injury as well."[101] The policy statement advised that while the term shaken baby syndrome "has its place in the popular vernacular," pediatricians should use the term "abusive head trauma" in their medical charts.[102] While the policy statement noted that medical diseases can mimic AHT and that pediatricians have a responsibility to consider alternative hypotheses, it did not identify the alternatives or offer any assistance in distinguishing between accidental, nonaccidental and natural causes, leaving this up to individual pediatricians.[103]

f. Increasing divergence. Given the disagreements between various organizations and the lack of consensus within organizations, it is increasingly difficult to gauge the extent to which doctors in general agree – or even have the knowledge needed to reach an informed decision – on whether abuse may be determined based on specific medical findings, or what those findings might be. In general, prosecutors and child abuse pediatricians continue to strongly endorse the SBS/AHT hypothesis, resulting in hundreds of successful prosecutions every year. At the same time, there is considerable discontent, particularly among forensic pathologists and

[98] Scientific Program, 40th Annual Meeting, National Association of Medical Examiners, San Antonio, TX (Oct. 13-18, 2006 (on file with authors).
[99] Deborah Tuerkheimer, *Science-Dependent Prosecution and the Problem of Epistemic Contingency: A Study of Shaken Baby Syndrome,* 62 ALA. L. REV. 512, 523-532 (2011).
[100] Christian, *supra* note 19.
[101] *Id.* at 1409.
[102] *Id.* at 1410.
[103] *Id.* at 1409-1410.

neuropathologists. For example, in a recent email, a forensic pathologist testifying on behalf of the prosecution in a criminal case advised the prosecutor that "I don't know what the breakdown is, but I would not be surprised to learn that it is close to 50/50 among neuropathologists, neurologists, and forensic pathologists as to whether any given case represents non-accidental trauma."[104] While this figure may be high, it seems clear that the consensus described by Dr. Narang is changing, and that there continues to be very little objective guidance on how to distinguish between accidental, nonaccidental and natural causes of findings previously viewed as diagnostic of shaking.

In 2012, the prediction of the dissenters in *Smith* that "it is unlikely that the prosecution's experts would today testify as adamantly as they did in 1997" may be coming to pass. In February 2012, in an Arizona post-conviction relief case, Dr. Norman Guthkelch, one of the first to hypothesize SBS, provided a declaration stating that the term "Shaken Baby Syndrome is an undesirable phrase and that there was not a vestige of proof when the name was suggested that shaking, and nothing else, caused the triad. Dr. Guthkelch went on to say that a number of other conditions – natural and non-accidental – may lead to the triad, including metabolic disorders, blood clotting disorders, and birth injury, to name a few. In the case at issue, he stated unequivocally that there was insufficient evidence to support a finding of homicide.[105] In the same case, Dr. A. L. Mosley, the medical examiner who conducted the autopsy and who previously testified that the cause of death was "Shaken/Impact Syndrome," stated that given the changes in the literature since 2000, there is no longer consensus in the medical community that the findings in his autopsy report are reliable proof of SBS or child abuse, and that if he were to testify today, he would testify that the child's death was likely due to a natural disease process, not SBS.[106]

Based on our own experiences, it appears that when subdural and/or retinal hemorrhages are present, child abuse pediatricians tend to

---

[104] E-mail from Mark Peters, MD, to Sharyl Eisenstein, Assistant State's Attorney, McHenry County, IL (Sept. 15, 2011) (on file with authors) (regarding Sophia Avila Case #08-073, which resulted in conviction, Oct. 14, 2011). In the same e-mail, Dr. Peters noted that infants can have a lucid interval of several days after head trauma and that a number of medical conditions can cause cerebral hemorrhage, retinal hemorrhage and bone fractures. These conditions should be ruled out before concluding that the injuries are the result of inflicted trauma. "Unfortunately, many or most, cannot be evaluated after death, and the pediatricians taking care of these children before death are not performing these tests for whatever reason. I am beginning to get the impression that when pediatricians see these kinds of cases, they see shaken baby or other non-accidental trauma right from the beginning (as evidenced in the dictated reports), and do not perform tests to rule out these other conditions." *Id.*

[105] Declaration of A. Norman Guthkelch, M.D., State of Arizona v. Drayton Shawn Witt, Feb. 3, 2012.

[106] Declaration of A. L. Mosley, M.D., State of Arizona v. Drayton Shawn Witt, Feb. 3, 2012.

diagnose child abuse (SBS/AHT) while forensic pathologists tend to diagnose blunt force trauma, with the manner of death categorized as accident, homicide or undetermined depending upon the circumstances of the case and the beliefs of the pathologist. While both groups recognize the overlap with natural causes, there is no commonly accepted protocol for investigating alternative causes and very little coordination with the relevant subspecialties. As the debate has turned increasingly harsh, moreover, clinicians outside the child abuse arena are often reluctant to participate in what may turn into a free-for-all in the courtroom and beyond.[107] Given this vacuum, many diagnoses and convictions continue to be based on the presumption that the triad or its components confirm abuse if the parents or caretakers cannot substantiate a known alternative.

g. The triad: where are we now? In 1996, it was generally accepted that, in the absence of a major motor vehicle accident or fall from a multistory building, the triad was caused primarily or exclusively by shaking. In 2001, we learned that the diffuse axonal injury attributed to shaking reflected hypoxia ischemia (lack of oxygen) rather than trauma, and that similar findings were found in infants who died natural deaths. By 2006, the "mimics" of SBS/AHT had expanded to include accidental trauma, birth trauma; congenital, genetic and metabolic disorders, infection, nutritional deficiencies, and a host of other conditions.[108] And in 2011, just five years later, a leading supporter of SBS theory stated publicly that "[n]o trained pediatrician thinks that subdural hemorrhage, retinal hemorrhage and encephalopathy equals abuse. The 'triad' is a myth."[109] As this suggests, we are dealing with an area that is far more complex and nuanced than previously recognized. We are, moreover, at the beginning, not the end, of our quest for evidence – a quest that requires much greater knowledge of the anatomy and physiology of the infant brain than is currently available. As we struggle to expand our knowledge, we need to engage in a careful and searching analysis of what went wrong while renewing our commitment to "getting it right."

C. Ongoing Debates

The debate over the validity of the SBS/AHT hypothesis has generated numerous subsidiary questions, including:

---

[107] The longstanding and coordinated attacks on those who disagree with the SBS hypothesis provide a strong deterrent for anyone who considers voicing a dissenting opinion. *See* notes 38 and 274 (with surrounding text).

[108] *See, e.g.* Sirotnak, *supra* note 10, at 193-214; *see also* Narang *supra* note 3, at 540 (differential diagnosis for subdural hemorrhages is extensive).

[109] Jenny, *supra* note 7, at slide 11.

1. Can short falls cause the triad, or is extreme force required?
2. Can there be a "lucid interval"?
3. What do retinal hemorrhages tell us about causation?
4. When do fractures, bruises or other features support an SBS/AHT diagnosis?
5. Do confessions confirm SBS/AHT?
6. How do we handle new hypotheses?

While these questions continue to produce vigorous and often acrimonious debate in the literature and the courtroom, there is sometimes surprising – and often under-recognized – consensus on key points.

1. ***Short falls.*** While it has long been recognized that short falls do not typically result in serious injury to young children,[110] it was understood for decades, if not centuries, that children sometimes suffered serious injury or death after falling short distances[111] and that the outcome of any given fall would be affected by a variety of biomechanical and physiological factors.[112] As mainstream medicine absorbed the SBS/AHT hypothesis, however, a new skepticism took hold that short falls could generate the force necessary to produce the triad. Since SBS/AHT theory held that such findings would require the force of a motor vehicle accident or multistory fall, the injuries attributed by parents and caretakers to short falls were automatically ascribed to abuse, typically violent shaking. New research has restored some of the traditional nuance as videotaped and witnessed short falls have confirmed that short falls can be fatal[113] and biomechanical studies have confirmed that the force of impact (including short falls) is much greater

---

[110] *See, e.g.*, Harvey Kravitz et al., *Accidental Falls from Elevated Surfaces in Infants from Birth to One Year of Age*, 44 PEDIATRICS 869, 873 (1969); Ray Helfer et al., *Injuries Resulting When Small Children Fall Out of Bed*, 60 PEDIATRICS 533 (1977); S. Levene & G. Bonfield, *Accidents on Hospital Wards*, 66 ARCHIVES OF DISEASE IN CHILDHOOD 1047 (1991); Thomas J. Lyons & R. Kim Oates, *Falling Out of Bed: A Relatively Benign Occurrence*, 92 PEDIATRICS 125 (1993).

[111] *See, e.g.*, John R. Hall et al., *The Mortality of Childhood Falls*, 29 J. OF TRAUMA 1273 (1989) (examining 18 pediatric deaths from falls under three feet, including two that occurred under medical observation). Falls from high chairs or changing table were similarly considered to be acceptable explanations for child deaths.

[112] *See, e.g.*, Barry Wilkins, *Head Injury – Abuse or Accident?*, 76 ARCHIVES OF DISEASE IN CHILDHOOD 393, 393, 395 (1997) (determinants of injury may include fall height; nature of surface; protective reflexes; whether fall is broken; whether child propelled himself; mass of body and head; proportion of energy absorbed, which may be influenced by which part of the body hits the ground first; whether some of the energy is dissipated in fractures; whether the contact is focal or diffuse; and whether there is secondary injury, including hypoxia/ischemia).

[113] *See, e.g.*, Plunkett, *supra* note 58; *supra* note 59; Hall, *supra* note 107; Paul Steinbok et al., *Early Hypodensity on Computed Tomographic Scan of the Brain in an Accidental Pediatric Head Injury*, 60 NEUROSURGERY 689 (2007) (reporting on radiology findings in five accidental fatalities, including a fall down stairs and a fall from a stool).

than the force of shaking.[114] The current consensus is that short falls (typically defined as falls of less than 3-4 feet) may occasionally cause death.[115]

The issues are therefore: how rare are short fall deaths, and how should this affect the interpretation of individual cases? Proponents of the SBS/AHT diagnosis often contend that, while short falls can be fatal, the chances are so remote as to be inconsequential.[116] In making this argument, supporters generally cite a 2008 article by Dr. Chadwick and Gina Bertocci that estimates the annual fatality rate for short falls among young children at less than one in a million.[117] To create a "best estimate" of the mortality rate, the authors selected a single injury database compiled by the State of California.[118] Like other epidemiological research, its reliability depends upon the accurate categorization of cases as "accidental" or "abusive." Since the time period of this data base (1997-2003) encompasses the peak of shaken baby theory, this data base may undercount short fall fatalities given the previously accepted belief that short falls could not kill.[119] In short, the data may reflect nothing more than the biases of the old understanding.[120]

---

[114] *See, e.g.,* Ommaya, *supra* note 78, at 226.

[115] John Plunkett & Mark Dias, Keynote Presentation, *Point/Counterpoint: Analysis of Outcomes from Short Falls,* Second International Conference on Pediatric Abusive Head Trauma (June 26, 2009) brochure at http://www.childdeathreview.org/Reports/2009PedAHTConference.pdf (Dr. Dias replaced Dr. Jenny, who was unavailable). *See also* David L. Chadwick, *Annual Risk of Death Resulting From Short Falls Among Young Children: Less than 1 in 1 Million,* 121 Pediatrics 1213 (2008) (13 short fall child fatalities listed in California data base, 6 of which the authors believe may be valid); Steinbok, *supra* note 109; Hall, *supra* note 107; Patrick E. Lantz & Daniel Couture, *Fatal Acute Intracranial Injury, Subdural Hematoma, and Retinal Hemorrhages Caused by Stairway Fall,* 56 J. OF FORENSIC SCIENCES 1648, 1649 (2011).

[116] David L. Chadwick, *Can a Short Fall Produce the Medical Findings of Shaken Baby Syndrome?,* NCSBS website, at http://www.dontshake.org/sbs.php?topNavID=3&subNavID=25&navID=278.

[117] Chadwick, *supra* note 111. Chadwick identifies three classes of cases that can be attributed to trauma: accident, (121 per million), homicide (22 per million) and short falls (0.48 per million). Even if these rates are correct, this would mean that 0.48 out of every 143.48 cases of traumatic fatal injury, or about one in 300, is attributable to short falls. Chadwick, *supra* note 111, at 1220. In the aggregate, nationwide, that would represent a significant number of incidents.

[118] Chadwick, *supra* note 111, at 1214, 1218. One study mentioned in Chadwick was discounted because the "fall histories [were] not validated" even though abuse had been ruled out by the police in all cases and two deaths had occurred under medical observation. *See* Hall, *supra* note 107, at 1274.

[119] The authors noted that the injury coding in the database often did not match the more detailed information in the death certificates. While the authors excluded cases incorrectly labeled as short fall deaths, they do not describe a corresponding effort to identify short fall deaths that may have been included in other categories, including homicide.

[120] This is another example of the circularity that affects much of the research in this field. If deaths presenting with the triad following a reported short fall are typically

Even if the Chadwick data is correct, however, it does not tell us whether any *particular* case is the result of accident or abuse. As Dr. Narang observes, "statistics embody averages, not individuals."[121] In individual cases, the issue is whether an injured child who appears in the emergency room after a reported short fall is suffering the consequences of a fall or is the victim of abuse. In this context, the Chadwick article is often cited to suggest that the likelihood that the death was attributable to the fall is less than one in a million.[122] In individual cases, however, it may be virtually certain that a short fall caused the injuries, *e.g.*, if the fall is confirmed by an independent witness or videotaped (as sometimes occurs with public surveillance equipment), even though the chances on average remain one in a million. More often, the medical evidence may confirm impact but cannot distinguish between a child who has fallen and hit his or her head and a child who has been hit on the head. The fact that fatal short falls are rare does not help us make this determination since child deaths are in and of themselves rare, and each cause (whether natural or accidental) is by definition even rarer.

In a large country such as the United States, moreover, small risks may translate into significant numbers. In 2010, there were approximately 12 million children under the age of 2 in the United States.[123] Using Chadwick's estimated mortality rate from short falls, one would expect perhaps 6 short fall deaths in the 0-2 age group. If a substantial number of short fall deaths in this age group were misclassified as SBS/AHT deaths based on the assumption that short falls could not kill, and if babies and toddlers are more vulnerable to short falls than older children, [124] these figures could increase substantially. This would be consistent with the biomechanical studies and case reports, which confirm that the forces generated by the types of short falls described in SBS/AHT cases (fall from parent's arms, fall down stairs, etc.) typically exceed accepted head injury criteria and may

---

diagnosed as SBS/AHT, the number of accidental short fall fatalities will appear to be vanishingly small. The rarity of short fall fatalities is then used to reject the caretaker's history of a short fall and to support an SBS/AHT diagnosis. This circularity issue is addressed below.

[121] Narang, *supra* note 3, at 522 (quoting JEROME GROOPMAN, HOW DOCTORS THINK (2007) at 6).

[122] *See, e.g.*, Brief for Plaintiff-Appellant at 6, State v. Louis, Wis. App. III Dist. No. 2009AP2502-CR , WL 4253604, ("[Y]es, a short fall could conceivably cause an infant's death, but it is exceedingly rare").

[123] The 2010 census recorded approximately 12 million children aged 0-2 in the U.S in 2010. Census Summary File 1, *Single Years of Age and Sex: 2010*, United States Census Bureau *at* http://factfinder2.census.gov/faces/tableservices/jsf/pages/productview.xhtml?pid=DEC_10_SF1_QTP2&prodType=table. Using Dr. Chadwick's estimate of 0.48 deaths per million children, the number of expected fatal short falls nationwide would be 5.76 (0.48 x 12) for children aged 0-2.

[124] Jenny, *supra* note 7, at slide 19 (overwhelming evidence shows that the response to a given injury in an infant is much worse than that of an adult to a similar injury).

be fatal.[125] Such deaths may be most likely to occur in children with pre-existing conditions, including chronic (old) subdural hemorrhages, coagulopathies (bleeding/clotting disorders) or pre-existing neurological impairment.

2. ***Timing ("lucid intervals").*** Under the traditional SBS/AHT hypothesis, it was believed that the child would be immediately unconscious upon infliction of the injuries, which were assumed to consist of ruptured veins and axons. The logical corollary was that whoever was with the child at the time of collapse must have inflicted the injuries.[126] This is, however, contrary to the well-known phenomenon of delayed deterioration from minor head injury, in which a prolonged period of normality or near normality may precede the collapse.[127] In 1998, Dr. Gilliland concluded that there was an interval of more than 24 hours (and sometimes up to 72 hours or more) between the trauma and the collapse in approximately 25% of alleged shaking, shaking impact or impact cases.[128] Subsequent studies and case reports have confirmed that collapse may not be immediate, even in cases involving impact.[129]

---

[125] *See* Jenny, *supra* note 79; Lantz, *supra* note 111.

[126] *See* Imwinkelried, *supra* note 36, at 5 ("In effect, the testimony time stamps the injuries, powerfully incriminating the last adult in the child's presence before the onset of symptoms").

[127] *See, e.g.,* J. W. Snoek et al., *Delayed Deterioration Following Mild Head Injury in Children,* 107 BRAIN 15 (1984) (reporting three delayed deaths in children apparently due to severe and uncontrollable unilateral or diffuse brain swelling). For this reason, hospitals typically urge parents and caretakers to monitor a child's symptoms after a head injury in order to detect delayed deterioration. *See, e.g.,* Head Injury guidelines, Seattle Children's Hospital, at http://www.seattlechildrens.org/medical-conditions/symptom-index/head-injury/ (directing parents to seek medical care immediately if child shows delayed neurological symptoms after head injury).

[128] M.G.F. Gilliland, *Interval Duration Between Injury and Severe Symptoms in Nonaccidental Head Trauma in Infants and Young Children,* 43 J. FOR. SCI. 723, 723 (1998).

[129] *See, e.g.,* Kristy B. Arbogast et al., *Initial Neurologic Presentation in Young Children Sustaining Inflicted and Unintentional Fatal Head Injuries,* 116 PEDIATRICS 180 (2005) (on rare occasions, infants or toddlers may sustain a fatal head injury yet present to hospital clinicians as lucid before death); Scott Denton & Darinka Mileusnic, *Delayed Sudden Death in an Infant Following an Accidental Fall, A Case Report with Review of the Literature,* 24 AM. J. FORENSIC MED. PATHOL. 371 (2003) (9-month-old acted normally for 72 hours after fall before fatal collapse); Robert Huntington, Letter, *Symptoms Following Head Injury,* 23 AM. J FORENSIC MED. PATHOL. 105 (2002) (reporting case of 13-month-old whose "severe intracranial injury symptoms...were delayed for several hours, during which time she was under our view and review in the hospital"). More recently, it has been noted that second impact syndrome – in which a minor impact occurring weeks to months after a more significant impact results in death – produces findings virtually identical to those in SBS/AHT cases. Robert C. Cantu & Alisa D. Gean, *Second-Impact Syndrome and a Small Subdural Hematoma: An Uncommon Catastrophic Result of Repetitive Head Injury with a Characteristic Imaging Appearance,* 27 J. OF NEUROTRAUMA 1557 (2010). This raises the possibility that the original trauma in some SBS/AHT cases may have occurred weeks to months before the collapse, possibly even at birth.

When the triad findings result from a natural disease process, the concept of a "lucid interval" may be meaningless because there may be no sudden precipitating event. Like any disease process, the natural mimics of abusive head trauma – ranging from stroke to metabolic or genetic disorders – may produce sudden and disastrous results, or may have a stuttering course, with a variety of warning signs and symptoms, followed by neurologic collapse. To determine the course of the disease, it is critical to obtain comprehensive and precise caretaker reports and to examine all records, including prenatal, birth and pediatric records. This information must then be coordinated with the radiology images, neurosurgical reports and/or tissue slides, which can provide objective information on cause and timing. Often, as one explores the child's history, it becomes apparent that multiple factors likely played a role in the collapse.

Today, there is no real dispute over whether lucid intervals can occur. Instead, the disputes about lucid intervals are more nuanced, usually arising over whether a lucid interval occurred in a particular case given the medical findings and symptoms. In a recent presentation, for example, Dr. Dias responded to the Gilliland research by noting that while children in the study experienced a period of lucidity following injury, all of the children who were seen by an independent observer "were described as *not normal*" during the interval.[130] However, the described symptoms, which included lethargy or fussiness, are signs of illness as well as head injury, and they provide little precision in timing.[131] Such symptoms are not infrequently noted in children diagnosed with SBS, suggesting that some of these children may be ill rather than abused.[132] Given these considerations, it has become increasingly difficult to time injuries or identify a perpetrator based on medical evidence alone.

3. ***Retinal hemorrhages.*** In recent years, the focus in SBS/AHT cases has shifted from subdural hemorrhages and brain swelling, which are known to have many causes, to retinal hemorrhages. For many years, ophthalmologists and pediatricians testified that in the absence of severe trauma, retinal hemorrhages were highly suggestive or even diagnostic of shaking.[133] This position is puzzling since retinal

---

[130] Dias, Presentation, *Concepts, Controversies and Conspiracy Theories in Abusive Head Trauma*, slide 12, New York City Abusive Head Trauma/Shaken Baby Syndrome Training Conference (Sept. 23, 2011) at http://www.queensda.org/SBS_Conference/SBC2011.html.

[131] Gilliland, *supra* note 124, at 724. *See also* Robert W. Huntington III, *Symptoms Following Head Injury*, 23 AM. J. FOR. MED. PATHOL. 105 (2002).

[132] *See, e.g.*, State v. Edmunds, 746 N.W. 2d 590, 592 (2008) (during the hours before her death, the child did not feed normally and cried inconsolably).

[133] *See, e.g.*, J.F. Geddes & John Plunkett, Letter, *The Evidence Base for Shaken Baby Syndrome*, 328 BRIT. MED. J. 719, 719 (2004) (many doctors consider retinal hemorrhages with specific characteristics to be pathognomonic of shaking; diagnosis is sometimes based on subdural or retinal hemorrhages alone).

hemorrhages are found in approximately one third of newborn babies[134] and in a wide range of conditions.[135] In adults, retinal hemorrhages are closely linked to intracranial hemorrhages in adults irrespective of cause, a phenomenon that is known as Terson syndrome.[136] To our knowledge, no explanation has ever been offered to explain why Terson syndrome would appear in adults but not in infants. Since infants are generally more vulnerable to illness or trauma than adults, one might suspect that, if anything, children would be *more* susceptible to retinal hemorrhage than adults.

Since it was clear by 2006 that children also develop retinal hemorrhage in a wide range of conditions,[137] supporters of the SBS/AHT hypothesis modified their claim that retinal hemorrhages are highly suggestive of abuse. Instead, they argued that certain variants – specifically, retinoschisis (separation of the layers of the retina), retinal folds (folding of the outer edges of the retina) and/or extensive retinal hemorrhages (retinal hemorrhages that affect many retinal layers and extend to the ora serrata) – are highly suggestive or even diagnostic of abuse.[138] In recent years, however, this hypothesis has also begun to unravel. Today, it appears that the size and scope of retinal hemorrhages may be largely associated with edema and time spent on life support rather than causation.[139] In addition, the severe, extensive retinal hemorrhages previously assumed to be diagnostic of SBS/AHT have also

---

[134] MV Emerson, *Incidence and Rate of Disappearance of Retinal Hemorrhage in Newborns*, 108 OPHTHALMOLOGY 36 (2001).

[135] *See, e.g.*, Narang, *supra* note 3, Appendices B and C; Patrick E. Lantz & Constance A. Stanton, *Postmortem Detection and Evaluation of Retinal Hemorrhages*, 12 PROC. OF THE AM. ACAD. FOR. SCI. 271 (2006) (retinal hemorrhages present at autopsy in infants who died from meningitis, asphyxia/suffocation, prematurity/congenital conditions, heart disease, in utero hemorrhage, blunt force trauma, sudden infant death syndrome/resuscitation, apnea/gastroesophageal reflux, and birth-related causes); Henry E. Aryen et al., *Retinal Hemorrhage and Pediatric Brain Injury: Etiology and Review of the Literature*, 12 J. OF CLINICAL NEUROSCIENCE 624 (2005) (retinal hemorrhages associated with an ever-expanding list of conditions). It has also, of course, long been known that retinal hemorrhages and, less commonly, cerebral edema are linked to the lack of oxygen at high altitudes. *See, e.g.*, Sankaranarayana P. Mahesh & Jeevan R. Mathura, Jr., *Retinal Hemorrhages Associated with High Altitude*, 362 N. ENGLAND J. MED. 1521 (2010); *see also* Fernando A. Moraga et al., *Acute Mountain Sickness in Children and Their Parents After Rapid Ascent to 3500 M (Putre, Chile)*; 19 WILDERNESS & ENVIRONMENTAL MEDICINE 287 (2008) (children more sensitive than adults to hypoxia from high altitudes).

[136] Albert Terson, *De l'hemorrhagie Dans le Corps Vitre au Cours de L'hemorrhagic Cerebrale*, 6 CLIN. OPTHALMOL. 309 (1900).

[137] *See* Lantz, *supra* note 131.

[138] *See, e.g.*, Narang, *supra* note 3, at 548-553, 557.

[139] Evan Matshes, *Retinal and Optic Nerve Sheath Hemorrhages Are Not Pathognomonic of Abusive Head Injury*, 16 PROC. OF THE AM. ACAD. FOR. SCI. 272 (2010) (retinal hemorrhages and optic nerve sheath damage may be linked to cerebral edema and advanced cardiac life support and are not limited to children who die of inflicted head injuries).

been identified in meningitis and an accidental short fall.[140] The Atlas of Forensic Histopathology summarizes the current state of knowledge on retinal hemorrhages as follows:

> The significance of retinal hemorrhage and optic nerve sheath hemorrhage is controversial. These hemorrhages are not, in and of themselves, sufficient to determine the presence of inflicted injury. Other circumstances under which retinal and optic nerve sheath hemorrhages may be found include resuscitation and cerebral edema. A recent retrospective study (Matshes, 2010) of 123 autopsies of children up to 3 years old showed retinal hemorrhage, optic nerve sheath hemorrhage, or both, in 18 cases. Of these, two were certified as natural deaths, eight as accidents, and eight as homicides. One finding of note was hemorrhage in six of seven cases without any head injury. There is a widespread belief among clinicians that skull fractures, subdural hematomas, and retinal hemorrhages do not occur in accidental short falls. In reality, all three have been found in cases of falls from short heights.[141]

In short, it is becoming increasingly unlikely that the size, shape or location of retinal or optic nerve sheath hemorrhages will prove to be an accurate indicator of abuse.

Retinoschisis and retinal folds are similarly no longer deemed virtually diagnostic (pathognomonic) of shaking or abuse. The traditional theory was that absent an automobile accident or the like, retinochisis or retinal folds could only be caused by the angular forces generated by the rapid acceleration and deceleration motion of shaking. However, a series of case reports has now established that retinoschisis and retinal folds also occur in accidental injuries that do not involve rapid acceleration/deceleration forces but instead involve other types of forces, such as crush forces. In one case a fourteen-month-old child suffered a skull fracture, subdural hematoma, and retinal folds when a television fell on him.[142] In another, a four-month-old child suffered a

---

[140] Juan Pablo Lopez et al., *Severe Retinal Hemorrhages in Infants with Aggressive Fatal Streptococcus Pneumonia Meningitis,* 14 J. AM. ASS. PED. OPHTHAL. STRAB. 97 (2010); Lantz, *supra* note 111, at 1648, 1649.

[141] PETER M. CUMMINGS, DARIN P. TRELKA & KIMBERLY M. SPRINGER, ATLAS OF FORENSIC HISTOPATHOLOGY 177 (2011); *see also* M. Vaughn Emerson, Elisabeth Jakobs & W. Richard Green, *Ocular Autopsy and Histopathologic Features of Child Abuse,* 114 OPHTHALMOLOGY 1384 (2007) (given our current lack of knowledge, much of what we think we know about the ocular findings of child abuse will continue to be the result of speculation rather than based on sound evidence).

[142] P. E. Lantz et al., *Perimacular Retinal Folds from Childhood Head Trauma,* 328 BR. MED. J. 754, 756 (2004).

fatal skull fracture with subdural hemorrhage and retinoschisis and retinal folds when a twelve-year-old child tripped and landed with her buttocks striking the infant's head.[143] In yet another case, a ten-week-old child suffered a skull fracture with subdural and subarachnoid hemorrhages, as well as retinal hemorrhages extending to the ora serata and retinal folds, when his mother, who was carrying him in a front-holding papoose, tripped and crushed his head between her chest and a wooden barrier.[144] Cases such as these have led researchers to conclude that, contrary to earlier beliefs, "there may be no retinal signs seen exclusively in non-accidental head injury."[145]

4. ***Bruises, fractures and other findings.*** In some cases, the triad is supplemented by bruises, fractures and other findings that can provide powerful confirmation of abuse. Ironically, however, such evidence may sometimes point in a different direction. While bruises are often taken as confirmation of abuse, particularly in infants, in whom bruises are unexpected,[146] Dr. Michael Laposata, one of the nation's leading coagulation experts, has pointed out that it is rarely possible to differentiate on external examination between bruises caused by trauma and those caused by coagulopathies (bleeding disorders).[147] While a child who presents with bruises, subdural hemorrhage and retinal hemorrhage may indeed be the victim of abuse and should be evaluated accordingly, it is important to be aware that these features are also consistent with genetic or acquired coagulopathies, including disseminated intravascular coagulation.[148]

---

[143] Gregg T. Lueder, Jane W. Turner & Robert Paschall, *Perimacular Retinal Folds Simulating Nonaccidental Injury in an Infant,* 124 ARCH. OPHTHALMOL. 1783 (2006).

[144] P. Watts & E. Obi, *Retinal Folds and Retinoschisis in Accidental and Non-Accidental Head Injury,* 22 NATURE 1514 (2008), available at http://www.nature.com/eye/journal/v22/n12/full/eye2008224a.html.

[145] *Id.* at 1.

[146] *See, e.g.,* Naomi F. Sugar, James A. Taylor, Kenneth W. Feldman and the Puget Sound Pediatric Research Network, *Bruises in Infants and Toddlers: Those Who Don't Cruise Rarely Bruise,* 153 ARCHIVES OF PEDIATRICS & ADOLESCENT MED. 399 (1999) ("Bruises are rare in normal infants and precruisers and become common among cruisers and walkers. Bruises in infants younger than 9 months and who are not yet beginning to ambulate should lead to consideration of abuse or illness as causative").

[147] Michael Laposata, Presentation, *Overdiagnosis of Child Abuse Due to Undiagnosed Underlying Disease,* Am. Assoc. of Clinical Chemistry Annual Meeting (December 2008) at *http://www.aacc.org/events/expert_access/2008/december/Documents/1208EA.pdf*; *See also* Martha E. Laposata & Michael Laposata, *Children with Signs of Abuse: When Is It Not Child Abuse?* 123 AM. J. CLIN. PATHOL., Supp. 1, S119, S120 (2005) (describing the "myriad of coagulopathies" that can mimic child abuse).

[148] *See, e.g., id.*; Marcel Levi & Hugo Ten Cate, *Disseminated Intravascular Coagulation,* 341 NEW ENGLAND J. OF MEDICINE 586, 586 (1999) (clinical conditions associated with disseminated intravascular association include sepsis, trauma, cancer, obstetrical complications, vascular disorders, reactions to toxins and immunological disorders).

Similar issues arise with skeletal findings. Contrary to popular belief, skull fractures may occur from birth trauma or household falls.[149] Other fractures or bony abnormalities may result from accidental trauma, metabolic bone disease and/or nutritional deficiencies.[150] In some cases, causation or vulnerability can be determined by testing and a careful medical history. In others, it may not be possible to differentiate between natural causes, accidental trauma and abuse on the basis of the medical findings alone.[151]

5. ***Confessions.*** As the differential diagnosis for the triad has expanded, the "case for shaking" as a mechanism of injury now rests largely on confessions.[152] SBS supporters argue that confessions prove that (a) some children with the triad were shaken; and (b) in the absence of a proven alternative, infants or children who present with the triad were almost certainly shaken.

The overriding problem is that confessions are not scientific evidence – and are rarely used as the basis for medical diagnoses – because the researcher cannot observe the underlying event. In the past decade, moreover, we have learned that confessions are not as reliable as

---

[149] *See, e.g.*, Brian C. Patonay & William R. Oliver, *Can Birth Trauma Be Confused for Abuse?* 55 J. OF FORENSIC SCIENCES 1123 (2010); Ross Reichard, *Birth Injury of the Cranium and Central Nervous System,* 18 BRAIN PATHOLOGY 565, 566 (2008) (incidence of skull fractures at birth is reported to be 2.9%); David S. Greenes & Sara A. Schutzman, *Occult Intracranial Injury in Infants,* 32 ANNALS OF EMERG. MED. 680, 684 (1998) (Duhaime reported that skull fractures were as likely to occur from falls of less than 4 feet as from falls of more than 4 feet, and that 18% of skull fractures in infants resulted from falls of less than 3 feet).

[150] *See* Kathy A. Keller & Patrick D. Barnes, *Rickets vs. Abuse: a National and International Epidemic,* 38 PEDIATR. RADIOL. 1210 (2008); Paul K. Kleinman, *Problems in the Diagnosis of Metaphyseal Fractures,* 38 PEDIATR. RADIOL. S388, S390-S392 (2008); Andrew Hosken, *Call For Vitamin D Infant Death Probe,* BBC RADIO 4 TODAY, Jan. 26, 2012 at http://www.bbc.co.uk/news/health-16726841 (parents acquitted of shaking child to death "after the jury learned that his fractures, supposedly telltale signs of abuse, could have been caused by his severe rickets....Michael Turner QC, who defended Miss Al-Alas, told the BBC that he was shocked by the lack of knowledge about vitamin D deficiency of some of the expert witnesses at the trial, held at the Old Bailey").

[151] *See* Alison M. Kemp et al., *Patterns of Skeletal Fractures in Child Abuse: Systematic Review*, 337 BRIT. MED. J. 1, 7 (2008) (no fracture on its own is diagnostic of child abuse); Carole Jenny, *Clinical Report: Evaluating Infants and Young Children with Multiple Fractures*, 118 PEDIATRICS 1299 (2006) ("[B]one diseases associated with increased bone fragility can be subtle or difficult to diagnose. These children are usually preverbal and cannot give a cogent history of their experiences. If abuse has occurred, caregivers of young children may not be forthcoming with a truthful history. On the other hand, family members of a child having an undiagnosed bone disorder may not be able to explain any mechanism of injury and may be completely bewildered by the injuries. Many parents of children with genetic or metabolic bone disease report that they were initially accused of abusing their children").

[152] *See, e.g.*, Dias, *supra* note 54, at 370 ("the consistent and repeated observation that confessed shaking results in stereotypical injuries that are so frequently encountered in AHT – and which are so extraordinarily rare following accidental/impact injuries – *is* the evidentiary basis for shaking") (emphasis in original).

once thought. Indeed, approximately 25% of the DNA exonerations in Innocence Network cases involved false confessions, guilty pleas or other incriminating statements to serious offenses such as rape and murder.[153] False confessions are produced in part by the psychological techniques used in interrogation,[154] including, among other things, the presentation of real or fabricated proof of guilt sufficient to make a suspect feel that the situation is hopeless.[155] An accused who is convinced that he or she will be convicted and believes that confessing will minimize the consequences (or at least put an end to the questioning) may well make a rational choice to confess, even falsely[156]—a type of confession recognized in the research literature as "coerced compliant false confessions."[157]

Confessions are particularly problematic in the child abuse area. First, there are remarkably few confessions – at least relatively few confessions that have been identified and examined in the research literature – relative to the large number of alleged shaking injuries (reportedly in the range of 1,200 to 1,500 per year in the United States).[158] One review of the child abuse literature from 1969 to 2001 found only 54 confessions to shaking, only 11 of which had no signs of impact.[159] As the author concluded, 11 cases (in this study,

---

[153] *False Confessions*, INNOCENCE PROJECT, http://www.innocenceproject.org/understand/False-Confessions.php (innocent defendants made incriminating statements, delivered outright confessions or pled guilty in about 25% of DNA exoneration cases). Indeed, in the Central Park jogger case, *multiple* defendants falsely confessed. *See, e.g., Anton McCray*, INNOCENCE PROJECT, http://www.innocenceproject.org/Content/Antron_McCray.php.

[154] *See* Mark Handler, Am. Assoc. of Police Polygraphists, PowerPoint Presentation, *Avoiding False Confessions and Defending Against Charges That You Obtained One* (2011) (on file with authors) (factors contributing to false confessions include investigator bias; pressure-filled interrogations; overconfidence on ability to tell truthful from deceptive subjects; certain coercive tactics; and context and subject characteristics that increase vulnerability).

[155] *See, e.g.*, Richard J. Ofshe & Richard A. Leo, *The Decision to Confess Falsely: Rational Choice and Irrational Action*, 74 DENV. U. L. REV. 979, 986 (1996-1997) ("investigators elicit confessions from the innocent...by leading them to believe that their situation, though unjust, is hopeless and will only be improved by confessing"); Steven A. Drizin & Richard A. Leo, *The Problem of False Confessions in the Post-DNA World*, N. C. L. REV. 891, 915 (2004) (the most effective technique used to persuade a suspect that his situation is hopeless is to confront him with seemingly objective and incontrovertible evidence of his guilt, whether or not any actually exists).

[156] Standard interrogation methods include cutting off denials of guilt and making the suspect believe that his situation is hopeless, followed by minimization strategies that present a confession as in his best interest. *See, e.g.*, Ofshe, *supra* note 151, at 998, 999.

[157] Ofshe, *supra* note 151, at 998.

[158] *See, e.g.*, NCSBS website at http://www.dontshake.org/sbs.php?topNavID=2&subNavID=10 (stating that an estimated 1,200 to 1,400 children are injured or killed by shaking every year in the United States); Tuerkheimer, *supra* note 37, at 10 (observing that an estimated 1,500 SBS diagnoses a year may provide "an outside parameter").

[159] Jan E. Leestma, *Case Analysis of Brain-Injured Admittedly Shaken Infants: 54 Cases, 1969-2001*, 26 AM. J. FORENSIC MED. PATHOL. 199, 199 (2005).

approximately 1 every 3 years on average) does not permit valid statistical analysis or provide support for many of the commonly stated aspects of shaken baby syndrome.[160] Three other articles – one in the U.S. and two in France – have addressed confessions to shaking but did not identify the confessions or the circumstances in which the confessions were obtained in sufficient detail to review their validity.[161] In two of these articles, moreover, the confessions did not reliably match the recorded medical findings, which included evidence of impact such as skull fractures, scalp swelling and bruising, underscoring the challenge with confessions.[162] In such cases, the confession may have understated the actions, or the shaking may have had nothing to do with the collapse.

Second, the definitions of "shaking" used in the literature and the courtroom are broad and ill-defined, and often include admissions to conduct that no one seriously argues could cause brain injury and death. As Professor Imwinkelried points out, Dr. Caffey's seminal 1972 article includes "burpings," a "confession" that a mother merely said "she and her husband 'might have shaken [the infant] when he cried at night,'" and a case in which a mother said she yanked a child to prevent him from falling off a bassinet onto the floor.[163] As Professor Imwinkelried noted, "[i]t is debatable whether such conduct should be characterized as the kind of major, violent shaking events that supposedly cause shaken baby syndrome."[164] In other cases, the confessions are to mild shaking intended to revive a comatose infant.[165] As Judge Posner of the U.S. Court of Appeals for the Seventh Circuit pointed out recently in *Aleman v. Village of Hanover Park*, this type of shaking is the proper way to initiate infant CPR; hence, admitting to it hardly constitutes a confession to deadly criminal abuse.[166]

Third, many of the confessions in child abuse cases involve interrogation techniques that are known to produce false confessions or plea bargains. Some interrogations include assertions that the medical

---

[160] *Id.* at 199.

[161] Suzanne P. Starling et al., *Analysis of Perpetrator Admissions to Inflicted Traumatic Brain Injury in Children*, 158 ARCH. PEDIATR. ADOLESC. MED. 454 (2004); Catherine Adamsbaum et al., *Abusive Head Trauma: Judicial Admissions Highlight Violent and Repetitive Shaking*, 126 PEDIATRICS 546 (2010); Matthieu Vinchon et al., *Confessed Abuse Versus Witnessed Accidents in Infants: Comparison of Clinical, Radiological, and Ophthalmological Data in Corroborated Cases*, 26 CHILDS NERV. SYST. 637 (2010).

[162] Starling, *supra* note 157, at 456; Adamsbaum, *supra* note 157, at 549.

[163] Imwinkelried, *supra* note 36, at 6 (quoting John Caffey, *On the Theory and Practice of Shaking Infants: Its Potential Residual Effects of Permanent Brain Damage and Mental Retardation*, 124 AMER. J. DIS. CHILD 161, 163 (1972)).

[164] *Id.* at 6, 7.

[165] *See, e.g.*, Aleman v. Village of Hanover Park, 662 F.3d 897 (7th Cir. 2011) (Posner, J.) (description of gentle shaking to elicit response from collapsed infant was interpreted as confession to violent shaking).

[166] *Id.* at 6.

evidence proves that a child was shaken and that only the accused could have done it. In *Aleman*, Judge Posner described such a scenario:

> They told him [the suspect] the only possible cause of Joshua's injuries was that he'd been shaken right before he collapsed; not being an expert in shaken-baby syndrome, Aleman could not deny the officers' false representation of medical opinion. And since he was the only person to have shaken Joshua immediately before Joshua's collapse, it was a logical necessity that he had been responsible for the child's death. Q.E.D. *A confession so induced is worthless as evidence, and as a premise for an arrest.*[167]

Sometimes these interrogation techniques may convince innocent parents or caretakers that they have committed a crime – a type of confession known in the research literature as "persuaded false confessions."[168] When confronted with "proof" of shaking or impact, parents may search their memories for what they might have done, ultimately recalling minor incidents that are then viewed as confessions or changing histories.[169] Some of these interrogations occur immediately after a child's death or serious injury, when distraught parents or caretakers may be particularly vulnerable to suggestion, manipulation or memory lapses.[170]

Other "confessions" are provided as part of a plea bargain. As elegantly described by Professor Tuerkheimer, acknowledgements of guilt accompanying a plea bargain may simply represent a cost-benefit analysis, with a full and logical evaluation of the circumstances.[171] Since innocent defendants charged with killing or severely injuring a baby confront a high likelihood that a jury will return a guilty verdict, a

---

[167] *Id.* at 16 (emphasis added); *see also* Emily Bazelon, *Shaken-Baby Syndrome Faces New Questions in Court*, N. Y. TIMES MAG. Feb. 2, 2011, at http://www.nytimes.com/2011/02/06/magazine/06baby-t.html?pagewanted=all (reporting the case of Dinesh Kumar, a Canadian father whose conviction was overturned after he had pled guilty to shaking his 5-week-old son to death; Kumar says that at the time of his guilty plea, he believed he had no hope of prevailing against the damning testimony of the state's pathologist, who has since been discredited for giving error-riddled testimony based on botched autopsies).

[168] *Id.* at 999 ("persuaded" false confessions "are given after a person has become convinced that it is more likely than not that he committed the crime, despite possessing no memory of having done so…[they] are elicited when an interrogator attacks and shatters a suspect's confidence in his memory"). These are known as internalized false confessions.

[169] *See, e.g.*, Aleman v. Village of Hanover Park, *supra* note 161.

[170] Research confirms that emotionally challenged individuals are more susceptible to the pressures and suggestiveness of interrogations. *See, e.g.*, Richard A. Leo & Deborah Davis, *From False Confession to Wrongful Conviction: Seven Psychological Processes*, 38 J. OF PSYCHIATRY & L. 9, 38-40 (2010).

[171] Tuerkheimer, supra note 95, at 532-535.

rational defendant who is offered a "substantial discount" will accept the terms of the offer, notwithstanding factual innocence.[172]

Finally, even if we assume that all shaking confessions are accurate and that shaking caused the collapse or death,[173] this still would not provide reliable evidence that the collapse or death in other cases was caused by shaking, any more than the confession of one bank robber to robbing a bank would provide reliable evidence that a defendant in another case was guilty of robbing a different bank. Today, we know that there are many alternative causes for findings previously attributed to shaking and that very few medical findings are specific for inflicted trauma. An assumption that shaking caused the collapse or death in cases with confessions would not, therefore, suggest that shaking caused the findings in cases without confessions.[174] At most, this would simply place shaking on the lengthy and ever increasing list of potential causes.

6. ***New hypotheses.*** In the past decade, researchers have struggled to differentiate between abuse, accidental trauma and natural causes. However, as Dr. Duhaime has pointed out, in this area, when you ask a question, you get an answer that more often than not leads to additional questions – a result that is very frustrating for those who want an answer and want it now.[175] Given the developments of the past decade, many more decades may pass – and many more hypotheses may be advanced and discarded – before we fully understand all of the causes of sudden infant death, with or without the triad. Today, we are still

---

[172] *Id.* at 534.

[173] This assumption is unlikely to be valid. For example, some shaking confessions occur in cases in which there is clear evidence of impact, including skull fractures and bruising. *See, e.g.*, Starling, *supra* note 157, at 456 (observing that 12% of "shaking only" confessions showed evidence of scalp or skull injuries). In other cases, the confession is to shaking around the time of the child's collapse, but the radiology and pathology establish that the injury was older. When the confessions do not match the injury, we do not know whether the confession was false or whether the shaking had nothing to do with the injuries, as in *Aleman*.

[174] Dr. Dias suggests that the "common and consistent admission by the perpetrator to shaking the infant . . . overwhelmingly suggests that shaking is an important component of infant abusive TBI and is, in fact, sufficient to cause the intracranial injuries found in AHT. To suggest otherwise (as required by the biomechanical evidence) would require that every confessed perpetrator has to have been consistently and universally lying about the same phenomenon, something that defies logic and common sense." Dias, *supra* note 54, at 369-370. However, the same analysis applies in the opposite direction: since most caretakers do not confess to shaking or any other form of abuse even when offered plea bargains but instead describe similar patterns, including short falls and/or sick or neurologically impaired babies, one would have to assume that these parents were consistently and universally lying about what they saw, a pattern that may indeed defy logic and common sense.

[175] Anne-Christian Duhaime, Cindy Christian & Susan Margulies, Presentation, *The Real Science: What Research is Telling Us about SBS/AHT: From Questions to Answers: Application of the Scientific Method to Abusive Head Trauma by Interdisciplinary Research Teams,* 11th International Conference on Shaken Baby Syndrome/Abusive Head Trauma Conference, National Center on Shaken Baby Syndrome (Sept. 12, 2010) (presentation notes on file with authors).

seeking answers to the questions that we have been asking for 40 years or longer – questions such as, why do some infants or toddlers suddenly collapse or die? Why do some of these children have subdural hemorrhages while others do not? What does the presence of the triad (or some elements of the triad) tell us about the cause of the collapse or death? And are there any findings that can accurately distinguish between accidents, abuse and natural causes? For decades, we thought we had answers to some of these questions: we thought that the presence of the triad, or some of its elements, proved that the child had been shaken. Today, the correct answer to these questions is, "we don't know." And, until we do know, we are, in Dr. Duhaime's words, simply "shooting in the dark."[176]

III. THE MEDICAL EVIDENCE: OLD AND NEW

Despite many warning signals, Dr. Narang argues that the research associating the triad, or some elements of the triad, with SBS/AHT is sufficiently reliable to form the basis for medical diagnoses and criminal convictions. While acknowledging that some of this research is marred by circularity,[177] he identifies a number of articles that he believes are sufficiently reliable to meet the standards of evidence-based medicine and *Daubert*. Dr. Narang further asserts that the biomechanical, neuropathological and anatomical research that casts doubt on the SBS/AHT diagnosis is unreliable and that the SBS/AHT diagnosis should rest on the judgment of clinicians, particularly child abuse pediatricians. In this section, we address each of these points.

A. Literature Supporting the AHT Diagnosis.

In the past decades, scores, if not hundreds, of medical articles have been published that examine the relationship between medical findings such as subdural and retinal hemorrhages and child abuse. Dr. Narang draws upon these studies to argue that highly significant statistical associations exist between subdural and retinal hemorrhages and child abuse, and that these associations are sufficient to support medical diagnoses of abuse and criminal convictions for assault or murder. While it is undeniable that a vast number of medical articles

---

[176] *Id.* at 14. In this remark, Dr. Duhaime was discussing the unilateral "big black brain," *i.e.*, the one-sided brain swelling found in approximately one-third of alleged SBS cases. Since shaking would be expected to damage both sides of the brain, the unilateral big black brain has always presented a pathophysiological conundrum. Anne-Christine Duhaime et al., Presentation, *The Real Science: What Research is Telling Us about SBS/AHT, From Questions to Answers: Application of the Scientific Method to Abusive Head Trauma by Interdisciplinary Research Teams*, Eleventh International Conference on Shaken Baby Syndrome/Abusive Head Trauma (Sept. 12, 2010) (notes on files with authors).

[177] Narang, *supra* note 3, at 560-561.

assert that their findings support the SBS/AHT hypothesis, this literature suffers from circularity and other methodological flaws. In this section, we describe the underlying methodology and its limitations, summarize the key studies, and identify some of the methodological and interpretive flaws that frequently appear in these studies.[178]

1. ***The methodology***. The studies cited by Dr. Narang follow the same basic methodology. In each study, the authors accept the basic premises of the SBS/AHT hypothesis and adopt criteria based on those premises to classify cases that present with subdural hemorrhage or other elements of the triad as accidental, abusive or natural. While the results of this classification vary depending on the precise criteria selected, the size of the sample and the sophistication of the analysis, each study found that if one adopts the SBS/AHT hypothesis, a relatively large percentage of cases resulted from abuse rather than accident. From these studies, Dr. Narang concludes that the presence of subdural and retinal hemorrhages is a statistically powerful indicator of abuse. This methodology does not, however, confirm the hypothesis or help us determine its validity. Nor does it tell us much about the diagnostic specificity of subdural and retinal hemorrhages. Instead, all that it tells us is what the resulting breakdowns would be if the hypothesis and the resulting classifications are correct.

This type of circular classification system can be used to "confirm" any hypothesis, irrespective of its validity. For example, one might hypothesize that dogs are by nature friendly and that they bite only if they have been abused or in pain. The logical corollary is that dogs that bite must have been abused or are in pain. If one adopts these hypotheses, dogs that bite but show no signs of pain must have been abused. The given history of "no abuse" would therefore be deemed inconsistent with biting, the owners would be assumed to be lying, and the dogs would be classified as "abused". If one further places into this category any dog who has ever bitten without evidence of pain, even as a puppy, the abuse rates for dogs might be extremely high, even approaching 100%. And the percentage of dogs for whom biting is a statistically reliable indicator of abuse would similarly be very high (theoretically 100%). This does not, however, confirm the hypothesis that biting dogs have been abused or that biting is statistically diagnostic of abuse; instead, it simply confirms what the breakdown would be *if* the hypothesis were correct. The abuse rates and correlation of biting to abuse might drop rapidly if one accepted alternative explanations, such as breed predisposition; age (very young or very old); instinctive protection of territory; poor eyesight; and/or fear of strangers.

[178] These studies largely address AHT as broadly defined, rather than SBS. Thus, even accepted at face value, they say nothing about the validity of shaking as the mechanism of injury and do not provide any support for the shaking hypothesis. As discussed below, because of methodological and interpretative problems, they also say relatively little about the causes and incidence of AHT.

In the SBS/AHT studies cited by Dr. Narang, the authors implicitly or explicitly accept the SBS/AHT hypothesis that subdural and retinal hemorrhages are generally traumatic in origin and require considerable force. The studies then use classification systems derived from this hypothesis to classify the findings as accidental, abusive or (in a few instances) natural. Thus, if the parent or caretaker describes a major accident, often characterized as equivalent to a motor vehicle accident or fall from a great height, the findings are classified as accidental. If the parent or caretaker cannot describe such an event – and particularly if the parent or caretaker describes a short fall or no trauma at all – the history is deemed to be inconsistent with the findings, and the case is classified as abusive. While some studies make an effort to eliminate natural causes, such as birth trauma, others do not. Overall, there is a general expectation that the parent or caretaker should be able to explain the medical findings – an expectation that is unrealistic in light of the broad range of causes.

2. ***The evidence.*** In the studies cited by Dr. Narang, the researchers typically select a cohort of children who have been diagnosed with head injury based on the presence of intracranial findings. Some studies focus on a particular element of the triad, such as subdural or retinal hemorrhage; others include evidence of impact, such as skull fractures or bruises. Using various criteria, the researchers then categorize the findings as abusive, accidental, natural or undetermined, with most studies attributing the findings to abuse if no known medical cause is found and the history is considered inadequate to explain the findings. The criteria for inadequacy vary considerably. For example, some researchers accept three-foot falls as a legitimate explanation for a subdural hemorrhage[179] while others accept only major motor vehicle accidents or falls from great heights.[180] Not surprisingly, the studies produce different breakdowns depending on the selection criteria, the sophistication of the analysis, and the inclusion of natural causes. The varying conclusions – producing abuse rates for subdural hemorrhages ranging from 28 percent[181] to 81 percent[182] in the studies discussed by Dr. Narang – are just one indication of the unreliability of "clinical

---

[179] Duhaime, *supra* note 43, at 179, 180 (intradural or subdural hemorrhages classified as neither presumptive nor suspicious for inflicted injury if the history is of a fall greater than or equal to three feet).

[180] Dimitra Tzioumi & R. Kim Oates, *Subdural Hematomas in Children Under 2 Years, Accidental or Inflicted? A 10-Year Experience,* 22 CHILD ABUSE & NEGLECT 1105, 1107 (1998) (motor vehicles accidents and falls from over eight feet considered sufficient to explain injuries).

[181] Jakob Matschke et al., *Nonaccidental Head Injury is the Most Common Cause of Subdural Bleeding in Infants <1 Year of Age*, 124 PEDIATRICS 1587 (2009).

[182] Duhaime, *supra* note 43, at 183. *Cf.* Alison M Kemp, *Abusive Head Trauma: Recognition and the Essential Investigation*, 96 ARCHIVES OF DISEASE IN CHILDHOOD EDUCATION & PRACTICE ED. 202, 205 (finding that "for a child under 3 years old with intracranial injury alone the probability of AHT was only 4%").

judgment" across hospitals, countries and time spans – the precise problem that evidence-based medicine and *Daubert* seek to address.

There are, however, common themes. Essentially, if natural causes are excluded or ignored (as is often the case) and if the outliers are removed, most studies find that approximately half (35 percent[183] to 60 percent[184]) of the parents or caretakers can provide an "acceptable" traumatic explanation for a subdural hemorrhage while approximately half cannot. Since the researchers generally assume that subdural hemorrhages require more force than other head injuries (including skull fractures), the "abuse" rate for subdural hemorrhages is typically much higher than the "abuse" rate for skull fractures and other head injuries.[185] This "abuse rate" is then used to confirm the high correlation between subdural hemorrhages and SBS/AHT.

In this section, we briefly describe the key findings in a selection of studies cited by Dr. Narang on subdural hemorrhages. We then discuss some of the methodological problems with these studies.

a. **Duhaime (1992).**[186] This study examined 100 consecutively admitted children 24 months of age or younger with a primary diagnosis of head injury. Subdural hemorrhages were classified as abusive if (i) they were accompanied by clinical or radiographic findings of focal impact with no history of trauma obtainable; (ii) the caregiver provided a history of a fall less than three feet when seen in association with a changing or developmentally incompatible history; *or* (iii) unexplained injuries such as healing long-bone fractures were present. Under this classification system, all of the subdural hematomas deemed accidental resulted from motor vehicle accidents; falls under three feet were categorized as trivial and constituted one prong of the test to confirm abuse.[187] There appears to have been no consideration of natural causes, including birth injuries. This study classified 81% of the subdural hemorrhages in the study group as abusive and 19% as accidental.

---

[183] Linda Ewing-Cobbs et al., *Neuroimaging, Physical, and Developmental Findings after Inflicted and Noninflicted Traumatic Brain Injury in Young Children*, 102 PEDIATRICS 300, 303 (1998).

[184] Kirsten Bechtel et al., *Characteristics that Distinguish Accidental from Abusive Head Trauma in Hospitalized Young Children with Head Trauma*, 114 PEDIATRICS 165, 176 (2004).

[185] For example, in 1992, Duhaime categorized 24% of head injuries and 81% of subdural hemorrhages as abusive. This same pattern is found in more recent studies. In 2005, for example, Vinchon classified 38% of head injuries and 64% of subdural hemorrhages as abusive. These and other studies are discussed below.

[186] Duhaime, *supra* note 43, at 179, 180, 181, 183.

[187] Consistent with Duhaime's earlier study (Duhaime, *supra* note 41), the authors concluded that shaking "does not generate sufficient deceleration forces" to cause subdural hemorrhages and brain injuries and that impact is required. They postulated that caretakers cause subdural hemorrhages by shaking, swinging or throwing the child, with the head stopping abruptly against a surface. No biomechanical or empirical support is provided for this hypothesis (Duhaime, *supra* note 43, at 183).

b. **Ewing-Cobbs (1998).**[188] This study examined 40 children ages one month to six years hospitalized for inflicted or noninflicted traumatic brain injury. In determining abuse, the authors used a classification scheme similar to that of Duhaime (1992) to determine whether a caretaker's history was compatible or incompatible with the findings. Head injuries were classified as abusive if the caretakers described falls of under four feet or from arm height. Children with documented prior histories of brain injury, metabolic/neurological disorders or prematurity (gestation of less than 32 weeks) were excluded from the study. This study categorized 64% of the subdural hemorrhages in the study group as abusive and 36% as accidental (most commonly in motor vehicle accidents).

c. **Feldman (2001).**[189] This study examined 66 children less than three years of age with subdural hemorrhages or effusions. Histories that were considered to be incompatible with the findings included all cases with no history of trauma; all short falls; stairway falls; and an adult falling on a child. The acceptable histories included motor vehicle accidents, falls from 10 feet or more, and major accidents (kicked by horse, dresser fell on head, and hit on head by falling log). Children with previously known hemorrhagic disease, previous neurosurgical procedure, previously recognized perinatal brain injury, meningitis, brain atrophy, central nervous system infections, renal dialysis, or severe dehydration/hypernatremia or cardiopulmonary bypass were excluded. This study categorized 59% of subdural hemorrhages in the study group as likely/highly likely/definite abuse; 23% as likely/highly likely/definite unintentional; and 18% as indeterminate.[190]

d. **Wells (2002).**[191] This study included 293 children less than three years of age with intracranial hemorrhages that were evident on radiological examination. Intracranial hemorrhages were categorized as abusive if (i) the caretaker offered no explanation for the findings, (ii) the findings were in the authors' view incompatible with the stated mechanism; or (iii) there was a confession of abuse. Children with a

---

[188] Ewing-Cobbs, *supra* note 179, at 301, 303, 307.

[189] Kenneth W. Feldman et al., *The Cause of Infant and Toddler Subdural Hemorrhage: A Prospective Study*, 108 PEDIATRICS 636, 637, 638, 639, 644 (2001).

[190] Histories considered indeterminate included a 2-month-old who fell from a kitchen counter onto a hardwood floor while restrained in a bouncy seat (minor injuries consistent with the fall but no independent witness); a fall by a father onto a 7-month-old with the father's full weight landing on the child (indeterminate since the mother was momentarily out of sight); a 2-month-old who fell down 3 carpeted stairs with his father (witnessed by maternal grandmother; child also had chronic effusions and rib fractures that could have been perinatal); and a 4-month-old who was in a truck that was hit by a crane, throwing the infant to the floor with his mother landing on top of him (child also had chronic effusions from possible birth injury).

[191] Robert G. Wells et al., *Intracranial Hemorrhage in Children Younger Than 3 Years*, 156 ARCH. PEDIATR. ADOLESC. MED. 252, 253, 254 (2002).

history of hemorrhage from prematurity, birth trauma, surgery or nontraumatic medical conditions were excluded. This study categorized 50.5% of intracranial hemorrhages as abusive, 37.2% as accidental, and 12.3% as undetermined.

e. **Bechtel (2004).**[192] This study examined 87 children under 24 months admitted with a diagnosis of head injury and who had a CT scan. Head injuries were categorized as abusive if (i) there was no history of a traumatic event (fall, blow to head or motor vehicle crash); (ii) the history of a traumatic event was incompatible with developmental level; (iii) the inflicted injury was witnessed; (iv) there was a confession; or (v) there were other physical injuries consistent only with inflicted injuries (e.g., pattern bruises, occult rib or extremity fractures). In this study, virtually all of the cases classified as abuse had no history of significant trauma. Natural causes and birth injury were not addressed. This study categorized 40% of subdural hemorrhages in the study group as abusive and 60% as accidental.

f. **Hobbs (2005).**[193] This study included 186 children less than two years of age with subdural hemorrhages from the United Kingdom and the Republic of Ireland. Causation was determined by reporting clinicians and pathologists without predetermined criteria. This study classified 57% of subdural hemorrhages as abusive, 30% as natural (perinatal, meningitis and other medical conditions), 9% as undetermined and 4% as accidental.

g. **Vinchon (2005).**[194] This study examined 150 children younger than 24 months of age hospitalized for craniocerebral traumatic lesions. The authors noted that the pathophysiology of subdural hemorrhages appeared to relate to the child's age rather than a specific cause of trauma. Twenty-one cases of birth trauma and five cases with natural causes (idiopathic macrocranium, hemophilia A) were identified. A disproportionate number of abuse cases had a history of perinatal illness (prematurity, obstructed labor, hospitalization after birth), which the authors speculated might have led to poor parental bonding. The authors did not appear to consider that these children may have been suffering from birth injuries.[195] This study classified 64.4% of subdural hemorrhages as abusive.

---

[192] Bechtel, *supra* note 180, at 166, 167.

[193] C Hobbs et al., *Subdural Haematoma and Effusion in Infancy: An Epidemiological Study*, 90 ARCH. DIS. CHILD. 952, 952 (2005).

[194] Matthieu Vinchon et al., *Accidental and Nonaccidental Head Injuries in Infants: A Prospective Study*, J. NEUROSURG: PEDIATRICS 380, 381, 383 (2005).

[195] Subdural hemorrhages, skull fractures, classical metaphyseal lesions (CMLs) and rib fractures may all be found at birth. *See, e.g.,* Rooks, *supra* note 91 (identifying subdural hemorrhages in nearly half of asymptomatic newborns); Rick R. van Rijn, *Birth-Related Mid Posterior Rib Fractures in Neonates: a Report of Three Cases (and a Possible Fourth Case) and a Review of the Literature*, 39 PEDIATRIC RADIOL. 30, 33 (2009) (fractures in full-term neonates are a well-known finding even after uneventful deliveries; CMLS and fractures of the clavicle, long bones, spine and skull have been

h. **Matschke (2009).**[196] This study looked at subdural hemorrhages in fifty autopsies of infants under one year of age. Since this study addressed children who died, it would have encompassed the most severe head injuries. At autopsy, 62% of the subdural hemorrhages were attributed to natural causes, 30% to trauma, and 8% to undetermined causes. The natural causes consisted of coagulation disorders (26%), perinatal conditions (26%), infection (8%) and metabolic disorders (2%). In a retrospective review, the authors classified the trauma cases as abusive if they resulted in a confession, criminal conviction, or at least three of the following findings: (i) subdural hemorrhage; (ii) retinal hemorrhage; (iii) an inadequate history; (iv) serious external injury, *i.e.,* hematomas or lacerations; (v) unexplained fractures of the long bones, ribs or skull; or (vi) simple or gliding contusions. Histories viewed as inadequate included sudden collapse/found lifeless; falls from a baby buggy, couch or father's arms; accidental head bumps; and, in one case, a confession of beating and shaking to stop crying. Under these criteria, all but one of the trauma cases were considered to be abusive. Thus, overall, 28% of the subdural hemorrhages were classified as abusive and 2% as accidental.

i. **Vinchon (2010).**[197] This study collected 412 cases of traumatic head injury in children under 24 months of age, classifying 30% of head injury cases as abusive and 70% as accidental. It did not separate subdural hemorrhage from other head injuries. Instead, it attempted to determine whether there were significant differences between confessed abuse cases and witnessed accidents. Forty-five cases of confessed inflicted head injury were compared with 39 cases of accidental trauma occurring in public places. The study found that 36.3% of the abuse cases (30 shaking, 15 beating) resulted in confessions obtained from judicial sources during or after the proceedings had been made public, as determined by a forensic pediatrician, while 13.5% of the accidents were corroborated by independent witnesses. In identifying SBS/AHT, the article endorsed the diagnostic value of what it called the "Ontario" triad, *i.e.,* subdural hemorrhage, retinal hemorrhage and no signs of impact,[198] rather than the classic triad of subdural hemorrhage, retinal hemorrhage and encephalopathy. In this series, clinical signs of encephalopathy were often minimal and brain

---

reported from birth trauma); Reichard, *supra* note 145, at 566 (incidence of skull fractures at birth is reported to be 2.9%).

[196] Matschke, *supra* note 177, at 1588, 1589, 1593.

[197] Vinchon, *supra* note 157, at 638, 639, 643, 644.

[198] The "Ontario" triad is based on an article by Michael Pollanen, Charles Smith and others. Charles Smith is the Ontario pathologist whose misdiagnosis of abuse in multiple cases in Ontario triggered the Goudge Inquiry. Michael S. Pollanen et al., *Fatal Child Abuse-Maltreatment Syndrome: A Retrospective Study in Ontario, Canada, 1990-1995,* 126 FORENSIC SCI. INT. 101 (2002).

ischemia was detected by CT scan in only 27% of abuse cases.[199] While the authors suggest that the use of confessions avoids the problem of circularity, it is difficult to assess this claim since the confessions were not spontaneous and there is no information on their content or the conditions under which they were obtained.[200] Based on confessions, the authors conclude that the presence of subdural hemorrhage, severe retinal hemorrhage and absence of impact provides "virtual certainty of abuse."

j. **Other studies.** Other studies cited by Dr. Narang use similar procedures to categorize cases as abusive, accidental or natural, with some considering a broader range of causes than others.[201] While fractures and bruises are often used to support findings of abuse, there is often relatively little effort to assess the age of these findings or to explore their relationship to nutritional deficiencies, coagulopathies or birth issues. Instead, most diagnoses of abuse continue to rest heavily on the inability of parents or caretakers to explain the medical findings – a process that is plagued with unknowns, even for medical professionals.

3. ***The flaws.*** As even a brief review of the literature suggests, the numerous studies that have concluded that SBS/AHT is a frequent cause of the triad and that subdural hematomas and retinal hemorrhages are reliable indicators of abuse have methodological flaws that range from circularity to statistical mishaps.

a. **Circularity**. The primary defect is that virtually all of the SBS/AHT literature is circular. In study after study, doctors assume that, in the absence of a known medical explanation, subdural hemorrhages are caused by major trauma. Cases are then classified as abusive if the parents cannot describe a major trauma or substantiate a natural cause. As set forth in articles by leading child abuse pediatricians, these criteria were still being used in 2008. For example, Dr. Reece proposed that when the triad was present, the diagnosis of SBS was "highly probable" when *one* of the following is present: no history of trauma; a history inconsistent with the injuries; a history that changes over time; witnessed shaking and/or impact; confession to shaking and/or impact; or additional information supplied by a multidisciplinary child-protection

---

[199] This study did not control for confounding variables, such as the evolution of the intracranial pathology in the interval between the injury and clinical assessment or scan, which was significantly different in the two groups of patients.

[200] The authors state that they had little data on the details, perpetrator, or mechanism of abuse. Under these conditions, it is impossible to verify causality or reliability (Vinchon, *supra* note 157, at 642).

[201] For example, a small study from Spain excluded 15 babies with subdural hemorrhages from birth trauma, accidental trauma, or natural causes, including CNS infections and glutaric acidosis. In the 20 remaining cases, the study identified 3 cerebrovascular accidents (2 arteriovenous malformations and 1 sinus thrombosis) and 2 coagulation disorders. Victoria Trenchs et al., *Subdural Haematomas and Physical Abuse in the First Two Years of Life*, 43 PEDIATR. NEUROSURG. 352, 353, 354 (2007).

team.[202] In a review, Dr. Hymel recommended omitting the second criterion (history inconsistent with the injuries) since that "presumes that we already know which histories are 'inconsistent' and which are 'consistent.'" Dr. Hymel suggested that additional research is needed to determine, with increasing precision, which histories are consistent and which are inconsistent.[203]

Under these standards, it is not surprising that some 50% of parents or caretakers cannot explain the findings to the satisfaction of the researchers. Contrary to Dr. Narang's suggestion, this does not prove that 50% of subdural hemorrhages are caused by abuse. All that it proves is that the researchers *believe* that this is so. One cannot validate a hypothesis based on a classification system that assumes the association that one wishes to prove. This is no different than deciding, *a priori,* that all male teenagers with long hair are drug users, assigning all male teenagers into "drug" and "drug-free" groups based on the length of their hair, and announcing that you have established a 100% correlation between long hair and drug use (and a corresponding 100% correlation between short hair and no drug use), with no effort to determine whether the correlation reflects reality.

Since the circularity problem is well-recognized – Dr. Jenny pointed it out in 2002 and Dr. Narang agrees that "some circularity is inevitable" – Dr. Narang asserts that "numerous well-designed studies [have] set out to control circularity in their experimental design."[204] For instance, in 2004, Bechtel[205] attempted to minimize circularity by using selection criteria based on "presenting history and physical examination findings."[206] As in other studies, however, "no history of traumatic event" was one of the criteria used to identify abuse,[207] with 12 of the 15 reportedly abused children characterized as abused based on this criterion.[208] Since there are many nontraumatic causes for subdural hemorrhages, this study almost certainly over-estimated the incidence of abuse.

Vinchon *et al.* later attempted to reduce circularity by examining cases of confessed abuse in France.[209] While this might seem to be a logical improvement over earlier studies, the reliability of confessions is far from certain, as discussed above. Not surprisingly, the greatest incentive and pressure to confess may occur when the doctors, investigators and judiciary believe that the triad is strong evidence of

---

[202] Robert M. Reece, *What Are We Trying to Measure? The Problems of Case Ascertainment*, 34 AM. J. PREV. MED. S117, S118 (2008).
[203] Kent P. Hymel, *Sample Review, Epidemiology,* QUARTERLY UPDATE at http://www.quarterlyupdate.org/epidemiology.
[204] Jenny, *supra* note 76, at 51-52; Narang, *supra* note 3, at 560-561.
[205] Bechtel, *supra* note 180.
[206] *Id.* at 166.
[207] *Id.*
[208] *Id.*
[209] Vinchon, *supra* note 157.

abuse since, in these cases, the alleged abusers will likely be told – not just by the doctors, police and prosecutor but often by their own attorneys and even their own families – that the medical evidence is conclusive and the hope for acquittal is slim to nonexistent. In such cases, the attorney may advise – and a parent or caretaker may realistically conclude – that the best option is to accept fault irrespective of guilt. In this study, the high rate of confessions (36.3%) combined with a lack of information on the cases and the fact that all confessions appear to have been obtained during judicial proceedings raises concerns with the reliability of the data.[210]

Other researchers, such as Matschke, attempted to address circularity by using criminal conviction as one of the inclusion criteria.[211] Since, however, such convictions are almost always based on the assumptions (and resulting medical opinions) that the research is designed to test, this criterion is entirely circular.

b. **Rule-out diagnoses.** In 1996, SBS was a "rule in" diagnosis, *i.e.*, if the triad elements were found, SBS was automatically diagnosed, at least in the absence of a known alternative cause. Today, SBS/AHT is a "rule out" diagnosis, *i.e.*, a diagnosis that can be made only if all other possible causes have been "ruled out" or excluded.[212] "Rule out" diagnoses are also known as diagnoses of exclusion or default diagnoses. By definition, these diagnoses occur when there is no laboratory test or direct evidence that would prove the diagnosis. If there were such a test or direct evidence, we would use them rather than going through the long, complex and ever-evolving list of "rule outs."

Because "rule out" diagnoses cannot be confirmed, they run a significant risk of being wrong. For example, doctors believed for years that stomach (gastric) ulcers were caused by stress: when they could find no other cause, the default diagnosis was that it must be the patient's fault.[213] As it turned out, however, ulcers are predominantly caused by bacterial infections.[214] Such misunderstandings of causation may do relatively little harm when there is no known treatment for the findings.[215] In contrast, misdiagnoses of child abuse cause immediate

---

[210] *Id* at 639.

[211] Matschke, *supra* note 177, at 1588.

[212] *See e.g.*, Jenny, *supra* note 7; Narang, *supra* note 3, at 569.

[213] *See, e.g.*, Press Release, *The Nobel Prize in Physiology or Medicine 2005, Barry J. Marshall, J. Robin Warren*, Nobel Prize website at http://www.nobelprize.org/nobel_prizes/medicine/laureates/2005/press.html (stating that stress and lifestyle were considered the major causes of peptic ulcer disease before the discovery of *Helicobacter pylori* by Barry Marshall and J. Robin Warren, who received the Nobel Prize for their work).

[214] *Id.*, *see also* Mayo Clinic Staff, *Peptic Ulcer: Definition*, Mayo Clinic website, at http://www.mayoclinic.com/health/peptic-ulcer/DS00242 (explaining that doctors now understand that bacterial infection or some medications, not stress or diet, cause most peptic ulcers).

[215] In the case of ulcers, one could argue that if an incorrect "rule out" diagnosis had not been propounded and widely accepted, the cause might have been discovered much

and often irrevocable harm by removing children from their homes, imprisoning innocent parents and caretakers, and destroying families. Such misdiagnoses may also result in improper or inadequate treatment for conditions that, if properly diagnosed, may have been eminently treatable.

The potential error rate of rule-out diagnoses increases as the number of alternative diagnoses expands. In SBS/AHT, there are tens or hundreds of known "rule outs," some of which can be identified only when the child is alive and others that can be identified only after death.[216] As described by Dr. Narang, the "rule-out" procedure requires a detailed whole body physical examination and complete medical history, including a detailed history of the complaints surrounding the presenting symptoms; any history of trauma, infection and/or exposure to infection; a detailed history of prior illnesses, surgeries and hospitalizations; birth history; developmental history; a history of relevant family medical illnesses/disorders; and a comprehensive psychosocial history.[217] In addition, the clinician must review the laboratory tests and radiology images and work with multiple agencies and medical specialties.[218] These findings then form the basis for a differential diagnosis, or list of possible causes. Dr. Narang suggests that many potential disorders can be eliminated through a detailed history, physical examination, and initial laboratory and radiologic results. In so doing, the clinician must synthesize the information gathered with "the known pathophysiologic processes of the human body, the evidence-based statistical information on the injuries, and the clinician's own experience in patient care."[219] This is a daunting task given the paucity

---

more quickly. The failure to identify the true cause of ulcers also resulted in unnecessary surgery that may have increased morbidity and mortality. *See, e.g.,* J. R. Todd Jr., *Peptic Ulcer Disease, an 11 Year Study*, 63 J. N. MED. ASS. 40, 43 (1971) (discussing morbidity and mortality rates following Billroth II procedures).

[216] For example, seizure activity and some coagulation abnormalities can only be identified when the child is alive, while slides of the brain and meninges, which may reveal congenital abnormalities or pre-existing injury, can only be obtained after death.

[217] Narang, *supra* note 3, at 569-571.

[218] *Id.* at 571; *see also* Jenny, *supra* note 7 (recommending an even more detailed "rule out" procedure which includes a complete evaluation of past history, including prenatal history; a family history going back generations, including unexpected deaths, genetic or metabolic disease; a social history; a complete systems review, including medications, allergies, immunizations and feeding history; a review of exposures, including travel, pets and toxins; a minute by minute "incredibly detailed" history of recent events; a detailed head to toe physical exam; a review of old records, including birth records, growth charts, past imaging studies, lab results and hospitalizations; extensive laboratory testing and radiology imaging, including MRI, MRA and MRV; and consults with specialists in many fields, including hematology, metabolic, genetics and infectious disease, as needed. For children who survive, the clinician should follow the child's long-term care; for those who do not, the clinician should attend the autopsy and consult with the medical examiner, as needed.)

[219] Narang, *supra* note 3, at 572.

of knowledge on the pathophysiology of the infant brain and the lack of evidence-based statistical information on causation. It is, moreover, unlikely that individual clinicians will have experience with the broad range of alternative causes, including childhood stroke and rare genetic conditions.

Despite the wide range of alternatives, Dr. Narang suggests that at the end of this process "in the vast majority of cases, the common denominator for SDH's and RH's will be trauma," in which case the clinician should distinguish between accidental and abusive head trauma by focusing on "inconsistencies."[220] Dr. Narang defines inconsistency as (i) the absence of a history; (ii) a history that substantially changes or evolves; (iii) a history that is inconsistent with the child's developmental capabilities; (iv) a history that is inconsistent with the pathophysiology of the injuries; or (v) a history that is inconsistent with the SBS/AHT literature. Dr. Narang concludes that in the presence of such inconsistencies, "the clinician can diagnose 'AHT/non-accidental trauma' with a reasonable degree of medical certainty."[221]

This process presents considerable challenges. For example, to determine if a particular injury is consistent with an accidental fall, the clinician must have a solid understanding of biomechanics and the unique characteristics of the fall; the unique characteristics and vulnerabilities of the child, including any genetic, nutritional or birth-related predisposing factors; the secondary metabolic response to injury; the anatomy of the developing brain; and the time course of the injury, including the impact of medical interventions.[222] Since there is strong evidence that an infant's response to a given injury is much worse than an adult's response to a similar injury,[223] what might appear to be minor or even trivial trauma in an adult may produce serious consequences in an infant, particularly one with predisposing conditions.[224] In looking at

---

[220] *Id.* at 572.

[221] *Id.*

[222] *See, e.g.,* Wilkins, *supra* note 108, at 393 (determinants of injury severity for a fall may include the distance fallen, the nature of the surface on to which the child falls, forwards or sideways protective reflexes, whether a fall is in some way "broken," whether the child propelled himself, the mass of the body and of the head, what proportion of the total kinetic energy is absorbed in compressing the ground and/or deforming the skull, brain or the rest of the body, whether the kinetic energy is dissipated in causing fractures, whether the contact with the ground is focal or diffuse, and the role of secondary brain injury such as hypoxic encephalopathy from an unprotected airway or ischemia from cerebral edema.)

[223] *See* Jenny, *supra* note 7 (there is overwhelming evidence that the response to a given injury in an infant is much worse than that of an adult to a similar injury).

[224] *See, e.g.,* Joseph H. Piatt, *A Pitfall in the Diagnosis of Child Abuse: External Hydrocephalus,*
Subdural Hematoma, and Retinal Hemorrhages, 7 NEUROSURG. FOCUS 4 (1999) (infants with external hydrocephalus may develop retinal and subdural hemorrhages spontaneously or from minor trauma); P.D. McNeely et al., *Subdural Hematomas in Infants with Benign Enlargement of the Subarachnoid Spaces Are Not Pathognomonic for Child Abuse,* 27 AM. J. NEURORADIOL. 1725 (2006) (subdural hematomas

the absence of a history or a history that substantially changes or evolves, moreover, the clinician must assess the possibility that the parent or caretaker truly does not know what happened to the child and that "changes" in the story may reflect improper interviewing techniques or the efforts of parents and caretakers to search their memories to help the doctors and investigators determine what happened to the child. To examine these factors, clinicians must evaluate the conditions under which the information was obtained, as well as the psychological condition of the caretakers.

Given the consequences of an abuse diagnosis, doctors must be just as careful – and just as knowledgeable – in weighing these considerations as in ordering major surgery or terminating life support, for in each and every case, they hold the future of a family in their hands. If, at the end of the analysis, the answer to whether particular injuries are accidental, natural or abusive is "we don't know," that is what needs to be said, and no more.

c. **Clinical judgment.** As Dr. Narang points out, it is not possible to conduct prospective randomized controlled studies in SBS/AHT research since it is not possible to violently shake babies for purposes of experimentation. Dr. Narang further points out that other medical diagnoses have not been validated by randomized controlled trials yet are widely accepted and uncontroversial.[225] For example, a doctor may listen to a patient describe symptoms that have been described as "migraine" and prescribe migraine treatment.[226] If the description of the symptoms accords with that of other migraine patients and the treatment works, the doctor may reasonably diagnose migraine based on clinical experience.

Doctors do not, however, have this type of clinical experience with SBS/AHT. In exercising clinical judgment, doctors generally correlate the patient's description of the symptoms and their onset (the patient history) with objective medical data (such as lab results) and response to treatment. Unlike a diagnosis of migraine, however, the SBS/AHT diagnosis is typically made in the context of patients who cannot talk, medical findings that lack definitive research, and a legal arena that demands near certainty (proof beyond a reasonable doubt). Since the parents or caretakers typically deny abuse, no one has seen it, and the infant obviously cannot verify it, there is no history to correlate with the findings.[227] There is similarly no course of treatment that would

---

may occur either spontaneously or as result of minor or unrecognized trauma in infants with benign enlargement of the subarachnoid spaces); Sirotnak, *supra* note 10, at 203 ("spontaneous or trauma-induced intracranial hemorrhages can occur in various common inherited coagulation disorders and those induced by another disease process or medical therapy").

[225] Narang, *supra* note 3, at 531-532.

[226] *Id.*

[227] One of the more unusual aspects of the SBS/AHT diagnosis is that clinicians typically reject the history provided by the caretakers and substitute their own

confirm or disprove SBS or AHT. Unlike a diagnosis of migraine, a diagnosis of intentional injury cannot be verified by response to a specific treatment or medication. With no history to correlate with the findings and no treatment that would confirm the diagnosis, the SBS/AHT diagnosis lacks the safeguards that gird most clinical diagnoses, including migraine.[228]

d. **Observer bias.** Observer bias refers to the innate cognitive biases that lead us to interpret data in ways that are consistent with what we expect to find.[229] Considerable research confirms that police investigators,[230] scientists,[231] and physicians[232] are all subject to cognitive errors that lead us to seek, recall, and interpret data in ways that support our initial judgments or hypotheses, and to disregard or minimize information that is inconsistent.

As reflected in the studies cited by Dr. Narang, cognitive biases are unavoidable when physicians use "clinical judgment" to determine which cases are abuse and which are accidental or natural. In *Hobbs*, for example, the authors acknowledged that "there is no absolute or gold standard by which to define NAHI [nonaccidental head injury]"[233] and declined to provide criteria for determining the causation of subdural bleeding. Instead, the authors deferred to the opinions of the treating physicians, who had been taught for decades that subdural hemorrhages in children were generally caused by abuse. Unsurprisingly, the treating physicians ascribed 57% of subdural hemorrhages and effusions to abuse. Even so, 57% is far from an overwhelming majority – far less

---

description of the events preceding admission, in effect creating a new patient history that then becomes the lynchpin of the diagnosis.

[228] As this suggests, SBS/AHT is not really a medical diagnosis but a legal conclusion. Doctors may reliably diagnose subdural hemorrhage, retinal hemorrhage and encephalopathy from radiology images and eye examinations. However, determining timing, causation and state of mind goes into areas that are more commonly reserved for pathologists, detectives, psychologists and juries.

[229] *See, e.g.,* D. Michael Risinger et al., *The* Daubert/Kumho *Implications of Observer Effects in Forensic Science: Hidden Problems of Expectation and Suggestion,* 90 CAL. L. REV. 3 (2002).

[230] Karl Ask & Pär Anders Granhag, *Motivational Bias in Criminal Investigators' Judgments of Witness Reliability*, 37 J. APPLIED SOC. PSYCH. 561 (2007); Karl Ask et al., *The "Elasticity" of Criminal Evidence: A Moderator of Investigator Bias,* 22 APP. COG. PSYCH. 1245 (2008); Keith A. Findley & Michael S. Scott, *The Multiple Dimensions of Tunnel Vision in Criminal Cases,* 2006 WIS. L. REV. 291.

[231] Andrea Follmer Greenhoot et al., *Prior Beliefs and Methodological Concepts in Scientific Reasoning*, 18 APPLIED COGNITIVE PSYCH. 203 (2004); Itiel E. Dror & David Charlton, *Why Experts Make Errors*, 56 J. OF FORENSIC IDENTIFICATION 600 (2006).

[232] Thomas S. Wallsten, *Physician and Medical Student Bias in Evaluating Diagnostic Information*, 1 MED. DECISION MAKING 145 (1981); Vicki R. LeBlanc et al., *Believing is Seeing: The Influence of a Diagnostic Hypothesis on the Interpretation of Clinical Features*, 77 ACADEMIC MED. S67 (Oct. Supplement 2002); Jesse M. Pines, *Profiles in Patient Safety: Confirmation Bias in Emergency Medicine*, 13 ACADEMIC EMERGENCY MED. 90 (2006); Mark L. Graber et al., *Diagnostic Error in Internal Medicine*, 165 ARCHIVES OF INTERNAL MED. 1493 (2005).

[233] Hobbs, *supra* note 189, at 954.

than the 81% identified by Duhaime and far below the criminal standard for proof beyond a reasonable doubt – making it difficult to apply these "statistics" in any given case.[234]

Similar disparities arose in a study in which 570 doctors (primarily pathologists and pediatricians) estimated the likelihood of abuse in 16 scenarios involving head injury.[235] In this study, the doctors were asked to classify the head injuries as unintentional, inflicted or undetermined.[236] While no case produced complete agreement, a majority opinion was considered achieved if more than 50% of all survey respondents and more than 50% of experienced respondents[237] rated the injury as either unintentional or inflicted. Using these standards, a majority opinion was achieved in only 8 of the 16 scenarios, 5 of which were classified as inflicted and 3 of which were classified as unintentional. In general, pediatricians were more likely than pathologists to classify cases as inflicted. As the authors noted, the inability to achieve consensus in 50% of the cases may be an appropriate recognition of the uncertainties that persist in this challenging arena.

Finally, observer bias influences the way in which we conduct research. To determine whether subdural or retinal hemorrhages are correlated with abuse, it is critical to determine whether and under what conditions these findings occur in children (or adults) who are not abused. Not surprisingly, the major scientific breakthroughs in SBS/AHT research have come through the examination of groups in which abuse is impossible or unlikely. Thus, from Geddes we learned that the swollen brains and thin subdural hemorrhages previously believed to be diagnostic of abuse are also found in infants who died from respiratory tract infection, perinatal asphyxia, gastroenteritis or sudden infant death syndrome (SIDS);[238] from Rooks we learned that thin subdural hemorrhages are present in 46% of asymptomatic newborns;[239] from Lantz, Matshes and Lopez we learned that retinal

---

[234] *Id.* at 952.

[235] Antoinette L. Laskey, Michael J. Sheridan & Kent P. Hymel, *Physicians' Initial Forensic Impressions of Hypothetical Cases of Pediatric Traumatic Brain Injury*, 31 CHILD ABUSE & NEGLECT 329 (2007).

[236] *Id.* at 332. Respondents classified the hypothetical cases into seven categories ranging from definitive unintentional to definitive inflicted, which were then collapsed into the three broad categories of unintentional, inflicted or undetermined by the study authors. ("In an effort to identify case examples of widely acceptable criteria for research definitions of unintentional and inflicted pediatric TBI, the participants' responses were collapsed from seven forensic categories into three, according to the following conservative schema: definitive or probable unintentional TBI were labeled unintentional; possible unintentional, undetermined, or possible inflicted TBI were labeled undetermined; and probable or definitive inflicted TBI were labeled inflicted").

[237] The study classified as experienced those physicians who indicated they had devoted 50% or more of their professional time to activities directly related to child abuse for at least 15 years. *Id.* at 332.

[238] Geddes, *supra* note 53, at 1304.

[239] Rooks, *supra* note 91.

hemorrhages are found in many types of deaths;[240] and from Holmes-Morton we learned that these findings may be associated with genetic abnormalities.[241] As this suggests, if we want to determine the full range of causes associated with the triad, we must go outside the child abuse arena and conduct studies that are free from observer bias and that look for the findings associated with abuse in children who collapse or die from natural causes.[242]

e. **Reversing the burden of proof.** Through a strange alchemy of legitimate confusion and flawed methodology, the burden of proof is reversed in SBS/AHT cases. The 2001 AAP Technical Report made the burden-shifting presumption explicit, stating that "data regarding the nature and frequency of head trauma consistently support the need for a presumption of child abuse when a child younger than 1 year has suffered an intracranial injury."[243] Once this presumption is in place, the burden is on the parents to "prove" an alternative explanation.

In so doing, Dr. Narang states that "[a] clear, biomechanically plausible account for how the injuries occurred should be available. When the history is absent, minimal, changing, or mechanistically implausible, suspicion of abusive injury is raised."[244] This standard raises two concerns. First, it assumes that the medical findings are traumatic and that doctors are able to accurately assess the biomechanical plausibility of the event. Second, in explaining the findings, parents are at a considerable disadvantage since they typically lack medical expertise and do not know what elements of the history might be important. Unlike doctors, moreover, who are encouraged to change their diagnoses as they acquire new information, parents are not

---

[240] Lantz, *supra* note 131; Lopez, *supra* note 136.

[241] *See, e.g.*, D. Morton Holmes et al., *Glutaric Acuduria Type I: A Common Cause of Episodic Encephalopathy and Spastic Paralysis in the Amish of Lancaster County, Pennsylvania,* 41 AMERICAN JOURNAL OF MEDICAL GENETICS 89 (1991); D. Holmes Morton, *Through My Window – Remarks at the 125th Year Celebration of Children's Hospital of Boston,* 94 PEDIATRICS 785 (1994); D. Holmes Morton et al., *Pediatric Medicine and the Genetic Disorders of the Amish and Mennonite People of Pennsylvania*, 121 AMERICAN J. OF MEDICAL GENETICS Part C 5 (2003).

[242] Since children who are asymptomatic or who are diagnosed with medical conditions do not routinely receive CT scans or eye examinations, we do not know the prevalence or characteristics of retinal and subdural hemorrhages in the general population or in specific medical conditions. We do know, however, that the more we look, the more we find. *See, e.g.*, Lantz, *supra* note 131; Matshes, *supra* note 135 (finding retinal hemorrhages in natural, accidental and abusive deaths); Lopez, *supra* note 136 (finding severe retinal hemorrhages in *Streptococcus pneumoniae* meningitis); Rooks, *supra* note 91 (finding subdural hemorrhages in 46% of asymptomatic newborns); Laura Rooms et al., *Hemophagocytic Lymphohistiocytosis Masquerading as Child Abuse: Presentation of Three Cases and Review of Central Nervous System Findings in Hemophagocytic Lymphohistiocytosis*, 111 PEDIATRICS e636 (2003) (reporting three cases of hemophagocytic lymphohistiocytosis initially misdiagnosed as suspected child abuse).

[243] Comm. on Child Abuse and Neglect, *supra* note 64, at 206.

[244] Narang, *supra* note 3, at 559.

permitted to add to the history as they learn more about the findings since this is viewed as a "changing story" and confirmation of abuse. This is especially problematic since the medical personnel and police often insist that the initial history cannot account for the injuries and pressure the caretaker to search his or her memories for additional details or other possible explanations. When the caretaker attempts to comply, however, any new details or possible explanations are viewed as a "changing story" and confirmation of abuse. Often, this is a circle from which there is no escape.

f. **Interpretive error: statistical misunderstandings.** Even if the studies cited by Dr. Narang and others did not suffer from circularity and other methodological flaws, they still would not provide a reliable statistical basis for diagnosing SBS/AHT. The statistical errors fall into two categories: misperceiving the significance of the *P-value*, and failing to avoid what is known as the Prosecutor's Fallacy.

(i) *P-value.* Dr. Narang claims that the studies he cites have tremendous statistical power because they achieve *P-values* of .05 or better. While that does indeed sound overwhelming, reliance on the *P-value* can be misleading. The *P-value* means that a finding is statistically significant based on the improbability that the conclusion attributed to a specific variable was caused by chance, using the standard threshold criterion of .05 (*i.e.*, the chance of a random rather than significant correlation is only 5%). The articles cited by Narang conclude that there is only a very small chance that the higher rates of subdural and retinal hemorrhage seen in cases involving abuse (as opposed to accidents or natural causes) are due to chance, indicating that the correlation is real rather than artificial (*i.e.*, produced by chance). Even if the causes were accurately classified, however, this measure provides no indication of the *strength* of the correlation for it does not distinguish between weak correlations (*e.g.*, subdural and/or retinal hemorrhages are 3% more likely in abuse cases than non-abuse) and strong ones (e.g., such findings are 80% more likely in abuse cases). Yet the strength of the correlation is precisely what is needed to satisfy fact finding requirements in criminal cases, which requires proof beyond a reasonable doubt. Statistical significance is necessary but not sufficient to support this evidentiary standard.

(ii) *The Prosecutor's Fallacy.* Dr. Narang's article makes a fundamental logical error that is so common that it has its own name: the Prosecutor's Fallacy.[245] It is the same mistake as saying: "Because

[245] *See* McDaniel v. Brown, 130 S. Ct. 665, 670 (2010); William C. Thompson & Edward L. Schumann, *Interpretation of Statistical Evidence in Criminal Trials: The Prosecutor's Fallacy and the Defense Attorney's Fallacy,* 11 LAW & HUM. BEHAV. 167 (1987); Michael I. Meyerson & William Meyerson, *Significant Statistics: The Unwitting Policy Making of Mathematically Ignorant Judges,* 37 PEPP. L. REV. 771, 778 (2010) (the "'prosecutor's fallacy' … incorrectly reverses events in a conditional probability to create a direct statement about the defendant's probability of guilt that is

lawyers tend to be literate people, literate people tend to be lawyers."[246] For example, Dr. Narang cites several studies for the proposition that AHT is more likely to cause subdural hematomas in infants than accidental trauma.[247] Even if these studies accurately assess causation, it would be an improper application of statistics to conclude that an infant who presents with a subdural hematoma is likely to have been abused.

Bayesian statistics teach that to determine the predictive value of an association – in this case, the likelihood that the presence of subdural or retinal hematomas indicates abuse – one must know not only the correlation between subdural hematoma and abuse but also the prior probability, or base rate, of abuse.[248] If the base rate of abuse is much smaller than the base rate of non-abuse, even an extraordinarily high correlation between subdural hematomas and abuse would not make abuse more likely than non-abuse when a child presents with a subdural hematoma.[249] Professor James Wood puts it this way: "Exactly the same evidence may lead to quite different conclusions, depending on the rate of abuse in the group being evaluated."[250]

A simple illustration makes this point. Suppose that an airport machine that checks for explosives hidden in checked bags is 99% accurate in detecting explosives. This means that the machine will sound an alarm 99 times if 100 bags with explosives are fed through the machine, and will sound an alarm only once if 100 bags without explosives are fed through the machine. In other words, bags containing explosives are 99 times as likely to make the alarm sound as bags not containing explosives. If the alarm sounds, how likely is it that the bag contains explosives? Probably not very likely at all. If one million bags are checked by machine, one of which contains explosives (a number that is almost certainly too high), there would be approximately 10,000 *false* alarms for every *true* alarm. By the same token, if the number of children with subdural hematomas from accidental or natural causes is significantly greater than the number with subdural hematomas from abuse, then Dr. Narang is wrong to assume from the studies he cites that subdural hematomas most likely indicate abuse.

The studies in Dr. Narang's article illustrate this point. In these studies, the correlation of subdural hematoma to abuse is very high but

---

not implied by the evidence. In logical reasoning, such an error is called "transposing the conditional") (footnotes omitted).

[246] Myerson, *supra* note 241, at 778.

[247] See above, pages 40-44.

[248] For a general overview of Bayesian statistics, see J. ARTHUR WOODWARD ET AL., INTRODUCTION TO LINEAR MODELS AND EXPERIMENTAL DESIGN 13-15 (1990).

[249] For a discussion of base rates, see James M. Wood, *Weighing Evidence in Sexual Abuse Evaluations: An Introduction to Bayes's Theorem*, 1 CHILD MALTREATMENT 25 (1996); Michael J. Saks & D. Michael Risinger, *Base rates, the Presumption of Guilt, Admissibility Rulings, and Erroneous Convictions*, 2003 MICH. ST. L. REV. 1051 (2003).

[250] Wood, *supra* note 245, at 26.

the base rate of abuse compared to non-abuse – to the extent it is revealed in the studies – is sometimes relatively modest, suggesting that subdural hematomas are at best only weakly diagnostic of abuse. Bechtel et al., for example, studied 82 children admitted for head trauma and concluded that 15 (18%) of the injuries were inflicted and 67 (82%) were "accidental."[251] Bechtel then reported that 80% (12/15) of the "inflicted" group had subdural hematomas while only 27% (18/67) in the "accidental" group had subdural hematomas.[252] From this, Dr. Narang concludes that, with a *P-value* of .001, "the association of SDH's with inflicted injury was highly statistically significant."[253] But that is only part of the story. When one factors in the low base rate of abuse, the conclusion is quite different. To compute the posterior probability of abuse, which more accurately reflects the diagnostic significance of subdural hematoma, one has to multiply the base rate by the likelihood ratio, which represents "the relative probability of coming across a particular piece of evidence in one group rather than in another."[254] Here, since 80% of purported inflicted cases have subdural hematomas and 27% of accidental cases have subdural hematomas, the likelihood ratio is 80:27, or 2.96:1. But because the base rate of abuse is only 18%, the true likelihood of abuse given subdural hematoma is only 35%.[255] One can make the same calculation in a different manner: since 18 of the subdural hematomas identified by Bechtel were accidental and 12 were inflicted, subdural hematomas were 50% more common in accident cases than in abuse cases. Either way, subdural hematoma is not diagnostic of abuse since most cases with this finding are non-abusive.[256]

A similar analysis applies to other studies. In the Matschke study, for example, the authors looked at 715 infant deaths, finding subdural hematomas in 50 of them.[257] Unlike the Bechtel study, the Matschke study attempted to identify all causes of the subdural hematomas, not just those attributed to trauma. Of the 50 cases with subdural hemorrhage, 15 (30%) were identified as traumatic and 35

---

[251] Bechtel, *supra* note 180, at 165.
[252] *Id.* at 167.
[253] Narang, *supra* note 3, at 544.
[254] Wood, *supra* note 245, at 26.
[255] The formula for computing the probability of abuse, also known as the posterior odds, using Bayes's theorem, is: Prior Odds (here, the base rate) x the Likelihood Ratio = Posterior Odds. *See* Wood, *supra* note 245, at 29. With prior odds (the base rate) of abuse of 1:5.56 (18%), and a likelihood ratio of 2.96:1, the posterior odds are: 1/1.56 x 2.96/1 = 2.96/1.56. That computes to a probability of abuse of about 35%, because converting odds into probability is accomplished by adding the numerator and the denominator of the odds together (2.96 plus 1.56 = 8.52) and dividing the numerator (2.96) by that total: 2.96/8.52 = .35 (35%). *See* Wood, *supra* note 245, at 28-29.
[256] The Bechtel study had only two classifications: inflicted or accidental. If some of the abuse cases were natural in origin, the base rate of inflicted abuse would have been even smaller.
[257] Matschke, *supra* note 177, at 1587.

(70%) were attributed to other causes.[258] Of the 35 cases that were not identified as traumatic, the subdural hemorrhages were attributed to bleeding/clotting disorders, perinatal events, infections, metabolic diseases, or (in 8% of the cases) undetermined causes.[259] A simple counting reveals that the study does not support the conclusion of its authors, which Dr. Narang quotes for the proposition that "most SDH's are attributable to trauma."[260] To the contrary, the data show that most SDH's are attributable to non-traumatic events, by a ratio of 70% to 30%.[261] As this suggests, while Dr. Narang is undoubtedly correct that some children who have been abused will have subdural hemorrhages, he commits the Prosecutor's Fallacy when he claims that children who have subdural hemorrhages are likely to have been abused. Instead, this is just one of many possible causes.

(iii) *Improper classifications.* These statistical misunderstandings assume even greater importance when superimposed on statistics that likely misclassify a significant number of medical findings as abusive. At present, we have no reliable statistics on the incidence of abusive head injuries. Instead, what we have are estimates of what the incidence would be if various hypotheses prove to be correct. Without some method of properly and accurately classifying the medical findings previously associated with shaking, there is no valid statistical basis for estimating the incidence of abusive head trauma in general, let alone the likelihood that abusive head trauma has occurred in specific cases.

B. The Skeptics: New Research, Old Anatomy

Two types of study cast doubt on the old SBS hypothesis: (1) studies that point out the lack of support for the traditional hypothesis, and (2) studies that identify specific problems with the hypothesis and/or suggest alternative causes. Dr. Narang dismisses both types of studies, suggesting that they were improperly conducted or are unsupported by the evidence.

1. ***Studies that identify the lack of support for the traditional SBS hypothesis.*** Dr. Narang focuses on Dr. Donohoe's 2003 study, "Evidence-Based Medicine and Shaken Baby Syndrome Part 1:

---

[258] *Id.* at 1587.
[259] *Id.* at 1589.
[260] Narang, *supra* note 3, at 542 (citing Matschke, *supra* note 177, at 1594).
[261] The Matschke study goes on claim that over 90% of the trauma cases were attributable to abuse. Matschke, *supra* note 177, at 1593. However, the study uses criteria that likely lead to an overestimation of the rate of abuse. *See* note 161, Matschke *supra* 177, at 1588, and related text. In any event, the study's conclusion that abuse is the most common cause of subdural bleeding in infants depends on dividing the natural causes into separate categories. If combined, they constitute 36% of cases, a greater proportion than that of alleged abuse.

Literature Review, 1966-1998," [262] which he dismisses as poor scholarship.[263] Specifically, he claims that Dr. Donohoe failed to capture the breadth of SBS/AHT medical research by using only the search term "shaken baby syndrome" in the Medline database and internet search.[264] Since, however, Dr. Donohoe was examining the evidence base for SBS, not for all types of traumatic brain injury, it was appropriate to search for articles using the phrase "shaken baby syndrome."[265] It was not until after Dr. Donohoe's analysis – and may have been partly as a result of his analysis – that the medical community began moving away from shaking as a mechanism and adopting more expansive terminology. Dr. Narang does not identify any research on shaking that Dr. Donohoe (or for that matter the participants in the 2002 NIH conference) missed. Without identifying the missing literature, Dr. Narang's criticism appears to be semantic rather than substantive.

Dr. Narang further criticizes Dr. Donohoe's observation that none of the SBS research achieved the "best evidence" standards of "Level 1," which includes randomized controlled trials.[266] We all agree that such studies are not possible since one cannot violently shake a child – let alone a large sample of children – to see what happens. Dr. Narang thus notes that "even the most ardent [evidence based medicine] advocate would admit that the best quality of evidence that can be expected in diagnostic studies is 'Level 2.'"[267] While Dr. Narang is correct that Level 1 evidence cannot be achieved in SBS research, this does not mean that Dr. Donohoe was incorrect to note that none of the SBS literature achieved Level 1 status and that none exceeded Level 3.[268] Instead, the lack of high quality evidence requires that clinicians and researchers exercise considerable caution in endorsing particular diagnoses or hypotheses, particularly when the adverse consequences are high. Rather than urging greater caution, however, Dr. Narang urges the courts to substitute the clinical judgment of pediatricians and others, which is by nature subjective, for the objective medical evidence envisioned by evidence-based medicine and *Daubert*. This suggestion

---

[262] Donohoe, *supra* note 82.

[263] Narang, *supra* note 3, at 533.

[264] *Id.* Dr. Narang contends that Dr. Donohoe should have searched for terms such as "Inflicted Neurotrauma,' 'Non-Accidental Trauma,' 'Whiplash Shaken Infant/Baby Syndrome,' or even more general terminology such as 'Subdural Hemorrhage/Hematoma' or 'Retinal Hemorrhage.'" *Id* at 533-534. Such expanded searches would have dramatically altered Dr. Donohoe's inquiry, broadening its scope far beyond his objective of identifying the research basis for shaken baby syndrome.

[265] Dr. Donohoe examined SBS research through 1998, a period in which SBS was an increasingly popular foundation for criminal convictions. As Dr. Donohoe observed, 1998/1999 is also regarded as "the turning point in acceptance of the tenets and practice of EBM [evidence based medicine]." Donohoe, *supra* note 82, at 239.

[266] Narang, *supra* note 3, at 534.

[267] *Id.*

[268] Donohoe, *supra* note 82, at 241 (by the end of 1998, no evidence on the subject of SBS exceeded QER III-2).

would lower the level of proof in child abuse cases and almost certainly result in mistaken diagnoses and false convictions – the very problems that evidence-based medicine and *Daubert* were attempting to address.

2. ***Studies that identify problems with the SBS/AHT hypothesis.*** Dr. Narang also criticizes studies that identify errors in the SBS literature, including the neuropathological studies conducted by Dr. Geddes and the more recent work on infant anatomy by Dr. Squier (a pediatric neuropathologist and a co-author), Dr. Mack (a pediatric radiologist) and Dr. Eastman (a clinical pathologist), claiming that this work is unsupported by the evidence. However, this research is extensively referenced to the medical literature. Once again, Dr. Narang does not identify any errors in the articles or the supporting literature.

In criticizing the work of Dr. Geddes, Dr. Narang selects his targets curiously. Dr. Narang does not discuss, or even mention, the groundbreaking research of Dr. Geddes and her colleagues in which they found that the brain swelling in alleged SBS/AHT cases was in most cases hypoxic-ischemic rather than traumatic, and that the subdural hemorrhages were typically thin, bilateral, and quite different in appearance from the traumatic hemorrhages found in older children and adults.[269] These observations, which are now generally accepted, called into question the traumatic origins of two of the three components of the SBS triad. Instead, Dr. Narang attacks Geddes III,[270] in which Dr. Geddes and her co-authors suggested a "Unified Hypothesis" to explain the mechanism of subdural hemorrhage and brain damage in allegedly abused infants. In Geddes III, the authors examined 50 non-traumatic infant deaths from infection, hypoxia and sudden infant death syndrome as well as three "shaken baby" deaths. Since all of the SBS deaths and most of the natural deaths showed intradural rather than subdural bleeding, the paper suggested that the mechanism might be vascular leakage from veins within the dura rather than the traumatic rupture of bridging veins. The paper further suggested that the intradural bleeding might result from a cascade of events combined with immaturity and hypoxia-induced vascular fragility.[271] Contrary to Dr. Narang's

---

[269] Geddes, *supra* note 53, at 1304 (observing that "axonal damage occurs in the brains of both head-injured subjects and in controls in much the same distribution…this is not 'DAI' [diffuse axonal injury]; but diffuse vascular or hypoxic-ischaemic injury, attributable to brain swelling and raised intracranial pressure"); Geddes, *supra* note 52, at 1297 (subdural hemorrhages found in cases of alleged non-accidental trauma are "materially different from those seen in adults, and are rarely 'massive'…They are almost invariably bilateral thin films of blood over the cerebral hemispheres, which do not require neurosurgical intervention").

[270] Geddes, *supra* note 89.

[271] *Id.* at 19 ("our observations in the present series indicate that, in the immature brain, hypoxia both alone and in combination with infection is sufficient to activate the pathophysiological cascade which culminates in altered vascular permeability and extravasation of blood within and under the dura. In the presence of brain swelling and raised intracranial pressure, vascular fragility and bleeding would be exacerbated by

assertion, Dr. Geddes did not recant this suggestion in her courtroom testimony but simply made clear that it was a hypothesis, akin to the SBS hypothesis, albeit more closely aligned with the anatomy of the infant brain.[272]

Like the Geddes studies, Squier and Mack's description of the "immature vascular plexus" is firmly rooted in anatomical research.[273] Indeed, this is an observational study of the kind described by Dr. Narang as "not just the norm but the cornerstone of medical diagnoses."[274] As Professor Goldsmith pointed out in 2001 and Dr. Reece pointed out in 2002, research on the physiology and pathophysiology of the central nervous system is essential to understanding the issues associated with SBS/AHT.[275] While Dr. Narang suggests that the existence of a highly vascularized immature dural plexus is simply a hypothesis, this description of the anatomy is based on microscopic examinations and resin casts, which are illustrated in the Squier and Mack articles.[276] Their descriptions are further confirmed by decades of anatomical research on the dura.[277]

---

additional haemodynamic forces, such as venous hypertension, and the effects of both sustained systemic arterial hypertension and episodic surges in blood pressure").

[272] In her testimony, Dr. Geddes stated that "[the 'unified hypothesis'] is not fact; it is hypothesis but, as I have already said, so is the traditional explanation.... [W]e do use the word "hypothesis" throughout [the paper]." R v Lorraine Harris, Raymond Charles Rock, Alan Barry Joseph Cherry, Michael Ian Faulder, 1 Cr App R 5, [2005] EWCA Crim 1980, Case Nos: 200403277, 200406902,200405573,200302848, at http://www.bailii.org/ew/cases/EWCA/Crim/2005/1980.html.

[273] Waney Squier & Julie Mack, *The Neuropathology of Infant Subdural Haemorrhage*, 187 FORENSIC SCI. INT. 6 (2009); Julie Mack, Waney Squier & James T. Eastman, *Anatomy and Development of the Meninges: Implications for Subdural Collections and CSF Circulation*, 39 PEDIATR RADIOL. 200 (2009).

[274] Narang, *supra* note 3, at 531-532.

[275] Goldsmith, *supra* note 55 ("Intimate collaboration is urged between biological specialists, medical professionals and biomechanicians to investigate crucial unsolved problems related to head injury, such as the rate of blood absorption from broken vessels by the body as a function of age, and the rate of effusion from ruptured vessels"); *Inflicted Childhood Neurotrauma*, *supra* note 66, at VIII ("[T]he contributions of basic scientists doing bench research related to the physiology and pathophysiology of the central nervous system are welcome and essential to the generation of understanding about these phenomena").

[276] Squier, *supra* note 269, at 8; Mack, *supra* note 269, at 203-205.

[277] *See, e.g.*, Erna Christensen, *Studies on Chronic Subdural Hematoma*, 19 ACTA PSYCHIATRICA ET NEUROLOGICA 69, 74 (1944) ("[t]he outermost fibrillary layer of the dura contains arteries as well as veins; the arteries are running in looping streaks, accompanied by two veins which open into the superior sagittal sinus. The arteries as well as the veins form anastomoses, the vessels branching dicotomically. Fine capillaries and arteries run obliquely through the dural tissue to the inner side where a nicely arranged, long-meshed capillary net is found, the junctions of which form ampullary blood-filled dilatations; and these ampullary dilatations constitute the connecting link between the capillary and venous systems. On the outer aspect a more wide-meshed capillary network is seen; and at the transition between the two capillary layers a few tiny vessels are seen"); J.A. Hannah, *The Aetiology of Subdural Hematoma: An Anatomical and Pathological Study*, 84 J. NERV. MENT. DIS. 169, 171

Squier and Mack further pointed out that the thin "subdural" bleeds traditionally associated with SBS/AHT in infants are unlikely to be caused by bridging vein rupture since the quantity of blood is too small given the volume of blood carried within these veins.[278] They also noted that there is no "subdural space", as hypothesized in traditional SBS theory; instead, the arachnoid and the dura are contiguous. Based on the anatomy, Squier and Mack observed that the blood-rich network of vessels in the inner layer of the immature dura may be the source of thin film bleeds found in infants, which are quite distinct from the thick, space-occupying subdural hemorrhages found in older children and adults. Dr. Narang does not identify any errors in these descriptions of the anatomy, which have been presented without objection at conferences on both sides of the debate.[279]

3. ***A shifting paradigm.*** Broadly speaking, the research dynamic between supporters and skeptics of the SBS/AHT hypothesis can be characterized as follows – supporters publish great quantities of research, in which selection criteria and clinical judgment based on the SBS/AHT hypothesis are used to differentiate abuse from accidents and natural causes. By failing to consider the wide range of known alternative causes or the unique pathophysiology of the infant brain, the

---

(1936) ("[c]ontrary to the usual conception, that the dura is a comparatively avascular structure, its blood supply is richer and much more complicated than would appear necessary to supply a structure, the functions of which are merely to support the brain and to act as an endosteum to the skull bones); C. W. Kerber & T.H. Newton, *The Macro and Microvasculature of the Dura Mater,* 6 NEURORADIO. 175, 179 (1973) (the dura contains "a vascular network which is complex and far in excess of the expected metabolic needs of a membrane furnishing only mechanical support); Hui Han et al., *The Dural Entrance of Cerebral Bridging Veins into the Superior Sagittal Sinus: an Anatomical Comparison between Cadavers and Digital Subtraction Angiography*, 49 NEURORADIO. 169 (2007).

[278] Squier, *supra* note 269, at 7-8 (rupture of the large caliber veins carrying large volumes of blood from the brain to the dural sinuses would be unlikely to produce the thin film haemorrhages characteristic of the young infant). The infant brain receives a large proportion of the cardiac output, creating substantial regional blood flow (averaging 40 ml/100 g per minute in a 6-month-old). The parasagittal bridging veins, which are strong and few in number, are responsible for draining a large proportion of the blood that flows through the supratentorial cortex. *Bilateral* subdural hemorrhages would require the rupture of multiple bridging veins, all of which would bleed at a relatively rapid rate. Since the bilateral thin film subdural hemorrhages in infants are typically small, sometimes no more than 5 cc, bridging vein rupture is an implausible explanation for these hemorrhages. *See also* Max Wintermark et al., *Brain Perfusion in Children: Evolution with Age Assessed by Quantitative Perfusion Computed Tomography*, 113 PEDIATRICS 1624 (2004).

[279] *See* Julie Mack, *Alternatives to Bridging Vein Rupture: Embryology and Function of the Infant Dura,* Presentation, EBMS Symposium (February 21, 2009) (brochure on file with authors); Waney Squier, Presentation, *The Pathology of Infant Subdural Hemorrhage and Brain Swelling*, EBMS Symposium (February 22, 2009) (brochure on file with authors); Julie Mack, Keynote Presentation, *The Dural Venous Plexus: Implications of Subdural Collections*, Second International Conference on Pediatric Abusive Head Trauma (June 26, 2009) brochure at http://www.childdeathreview.org/Reports/2009PedAHTConference.pdf.

studies almost certainly overestimate the incidence of abuse. Dr. Narang aggregates this data and presents it as persuasive statistical evidence that subdural and retinal hemorrhages are reliable indicators of abuse. In making these claims, Dr. Narang also fails to consider the base rates of abuse and non-abuse when making statistical claims about the diagnostic power of subdural and retinal hemorrhages. Nonetheless, irrespective of its evidentiary basis and statistical validity, the sheer volume of this research serves to intimidate those who are not familiar with its methodological shortcomings.

At the same time, researchers and clinicians who question the SBS/AHT hypothesis or suggest alternatives based on biomechanical studies or the anatomy of the infant brain routinely confront personal and professional attacks on their motivation, competence and integrity.[280] These attacks have slowed the research and deterred others from addressing these important issues.[281] What Dr. Narang and other supporters of the SBS/AHT hypothesis fail to mention, however, is that despite these vociferous attacks, most of the work that they have attacked in the past has been absorbed into the mainstream, slowly but certainly shifting the paradigm. As this suggests, the recent changes in terminology are not semantic but instead reflect the slow process of discarding previous "truths" about SBS.

At present, the new paradigm includes general agreement on the following points:

- Subdural hemorrhages in infants are not caused exclusively or almost exclusively by shaking or inflicted trauma.
- The dura is far more complex than previously understood, with some hemorrhages previously identified as subdural arising within the dura.
- Thin subdural hemorrhages are found in nearly half of asymptomatic newborns, confirming that they are not always symptomatic and can occur without brain damage.
- Rebleeds of chronic subdural hematomas can and do occur.

---

[280] These attacks appear to be largely coordinated by the NCSBS. *See, e.g.*, Holmgren, *supra* note 39 (Pinocchio slides and sing-along); Colin Welsh, Presentation, *A National Co-ordinated Approach to Cases of Non-Accidental Head Injury in the UK*, 11th International Conference on Shaken Baby Syndrome, sponsored by the National Center on Shaken Baby Syndrome (Sept. 2010) (describing efforts of New Scotland Yard and child abuse prosecutors to silence experts who question the diagnosis) (notes on file with authors); Brian K. Holmgren, *Irresponsible Expert Testimony*, NCSBS website at http://dontshake.org/sbs.php?topNavID=3&subNavID=28&subnav_1=96&navID=115.

[281] In a recent discussion of an SBS case on the Fifth Estate, a Canadian investigative program, a defense attorney said that he had talked to 50-60 experts who questioned SBS theory, but that only two were willing to testify for fear of being blackballed. Television Program, *Diagnosis Murder*, THE FIFTH ESTATE (January 13, 2012) available at http://www.cbc.ca/fifth/2011-2012/diagnosismurder/.

- Retinal hemorrhages are not caused exclusively or almost exclusively by shaking or other forms of trauma.
- Retinal folds and retinoschisis are not diagnostic of abuse.
- The brain swelling in alleged SBS/AHT cases is hypoxic-ischemic rather than traumatic.
- Impact, even on a padded surface, generates more force than shaking.
- Short falls can present with the triad and result in death.
- Lucid intervals can occur in trauma cases.
- The concept of a lucid interval does not apply when the triad arises from natural causes.
- There is a long list of alternative causes for the triad, ranging from birth trauma to genetic abnormalities, infection and childhood stroke.

As the new paradigm emerges, new cases must be evaluated – and old cases re-evaluated – with the same commitment to meticulous diagnosis found in any other complex area of medicine. Our understanding of the medicine and the biomechanics of injury must be combined with a recognition that many fundamental questions remain unanswered. In the meantime, we must strive to make the best possible decisions under conditions of uncertainty—conditions that require us to balance the unthinkable harm of child abuse against the equally unthinkable harm of destroying families and imprisoning innocent parents and caretakers based on a flawed hypothesis.

To this end, in 2011 two of our co-authors – Dr. Barnes and Dr. Squier – published invited reviews of the literature in their own areas of expertise, pediatric neuroradiology and pediatric neuropathology. These reviews describe our current state of knowledge on the medical findings previously attributed to shaking as well as the ever-expanding list of alternative diagnoses.[282]

IV. MEDICAL AND LEGAL STANDARDS OF RELIABILITY

While we now have a better understanding of potential causes for subdural hemorrhage, retinal hemorrhage and encephalopathy, the issue has become: how much of this evidence is sufficiently reliable for medical diagnosis and courtroom testimony?

A. Medical Diagnosis: Art or Science?

As Dr. Narang recognizes, there has been a shift in medicine towards the objective examination of the quality of the evidence supporting established theories. The movement known as evidence based

[282] Barnes, *supra* note 12; Squier, *supra* note 12.

medicine represents an effort to examine the reliability of the evidence on which doctors make diagnoses and order treatment.[283]

Under the standards of evidence-based medicine, clinicians formulate questions, conduct literature searches to identify the best available evidence, and critically assess the reliability of that evidence.[284] In so doing, clinicians need to distinguish high from low quality primary studies, identify knowledge gaps and frame questions to fill those gaps, and apply the research evidence to the particular patient.[285] Evidence-based medicine guidelines assist in this process by providing a hierarchy of evidence, ranging from randomized controlled trials to unsystematic clinical observations.[286]

While randomized controlled trials of child abuse are not possible, a review of the literature indicates that the problem goes much deeper: the real problem is that the literature cited in support of the SBS/AHT hypothesis falls at the bottom of the hierarchy of evidence and rests almost entirely on assumptions and hypotheses, combined with emotionally compelling demonstrations and anecdotal evidence, largely in the form of confessions. Recent research has made clear that many of the underlying assumptions are inconsistent with the anatomy and physiology of the infant brain.

To address the lack of an objective evidence base for the SBS/AHT hypothesis, Dr. Narang recommends that the clinical judgment of child abuse pediatricians be substituted for evidence-based medicine. This proposal circles back, however, to the original problem: even the most popular clinical judgments can be wrong, as evidenced by a long list of misguided clinical judgments, ranging from lobotomies to ulcers to hormone replacement therapy.[287] Organizational acceptance of clinical judgments is not, moreover, persuasive. As Daniel Kahneman, the Nobel Prize winning Professor of Psychology and Public Affairs at Princeton University, points out, this problem is not unique to medicine:

---

[283] *See, e.g.*, David L. Sackett et al., *Evidence Based Medicine: What It Is and What It Isn't*, 312 BRIT. MED. J. 71, 71 (1996) ("[e]vidence based medicine is the conscientious, explicit, and judicious use of current best evidence in making decisions about the care of individual patients); Frank Davidoff et al., *Evidence Based Medicine*, 310 BRIT. MED. J. 1085, 1085 (1995) ("clinical decisions [in evidence based medicine] should be based on the best available scientific evidence...and the clinical problem – rather than habits or protocols – should determine the type of evidence to be sought").

[284] *Id.; see also* Robert C. Hawkins, *The Evidence Based Medicine Approach to Diagnostic Testing: Practicalities and Limitations*, 26 CLIN. BIOCHEM. REV. 7 (2005); Guyatt, *supra* note 1, at 1290-1296.

[285] *See* Guyatt, *supra* note 1, at 1290, 1293 (clinicians should seek evidence from as high in the appropriate hierarchy of evidence as possible and apply it to the particular circumstances of the patient); Hawkins, *supra* note 280, at 8 (clinicians must determine whether the research used independent reference standards and was applied to a population of patients comparable to the patient in question).

[286] Guyatt, *supra* note 1, at 1292; *see also* Phillips, *supra* note 74.

[287] *See, e.g.*, Guyatt, *supra* note 1, at 1293 (hormone replacement therapy does not help prevent coronary artery disease despite several observational studies that had shown "dramatically positive results").

history has shown that "people can maintain an unshakeable faith in any proposition, however absurd, when they are sustained by a community of like-minded individuals."[288] In this case, the reluctance to apply the standards of evidence-based medicine to SBS/AHT has been exacerbated by the efforts of advocacy groups dedicated to the promulgation of the SBS/AHT hypothesis and the criminal prosecution of SBS/AHT cases.[289] While we support their commitment to the prevention of child abuse, this commitment should not substitute subjective beliefs for objective scientific evidence. Instead, the commitment must be to getting it right.

Given the current state of knowledge, what is it reasonable for medical personnel to suggest? Is this simply one of the areas in which "the evidence is so sparse, that EBM simply cannot be instructive either for Medicine or Law"?[290] The answer to this question depends on the facts of the case and the proposed solutions. SBS/AHT cases range from cases with obvious head trauma (facial bruising, skull fracture and/or soft tissue swelling) to cases in which seemingly healthy children have suddenly and inexplicably collapsed. Sometimes the history and a meticulous review of the medical records provide a likely answer; other times, it is not possible to determine causation based solely on the medical evidence.

In the face of such uncertainty, we must look closely at the costs and benefits of the proposed solutions. The answers are simplest when we are dealing with prevention. Because violent shaking is dangerous and has no known benefits, there are few costs and many potential benefits associated with educating parents that they should never shake a child. Because short falls can be fatal, parents should also be warned that children should not be placed on counters or couches, or in other places from which they might fall or where other children or adults might fall on them.

Similar principles apply to treatment. Because the body cannot always distinguish between trauma and illness, we need to constantly examine and re-examine our treatment protocols to ensure that we are providing the best possible care to children who present with the triad or one of its components. If the head findings are primary, we need to be able to quickly and accurately distinguish between the various possibilities (e.g., injury, infection or stroke) so that we can provide appropriate treatment. If the head findings are secondary, we need to promptly identify and treat the underlying illness or condition if the child is to survive.

---

[288] DANIEL KAHNEMAN, THINKING, FAST AND SLOW 217 (2011).

[289] Of these, the most prominent is the NCSBS, which since the 1990s has taken a lead role in training prosecutors, doctors and social workers. Active participants in the NCSBS have been involved in the NAME and AAP policy statements and the more recent certification of child abuse pediatricians.

[290] Narang, *supra* note 3, at 521-522.

The burden shifts when the solution is to destroy families and imprison parents. Based on what we now know, it is inappropriate for medical professionals to diagnose shaking or abusive head trauma based solely or primarily on the presence of subdural hemorrhage, retinal hemorrhage and/or encephalopathy. When a child abuse referral or diagnosis is made based on these findings, it should be clearly disclosed that there are many possible causes for these findings; that the issues are complex and poorly understood; and that an SBS/AHT diagnosis based exclusively or primarily on these findings rests on good-faith beliefs and hypotheses, rather than science.

B. Daubert: Is SBS/AHT Ready for the Courtroom?

As Dr. Narang states, in determining reliability for admissibility purposes under *Daubert*, courts may consider: (1) whether a theory or technique can be (and has been) tested (also known as falsifiability or testability); (2) whether the theory or technique has been subject to peer review and publication; (3) whether there is a known or potential error rate; and (4) whether there is general acceptance in the relevant scientific community.[291] In addition, the courts must consider whether the theory is "tied sufficiently to the facts of the case."[292]

Dr. Narang does not argue that the medical literature on SBS/AHT meets the technical standards of *Daubert* (particularly factors 1 and 3) but argues that the courts should instead accept the "clinical judgment" of doctors, particularly child abuse pediatricians, that abuse has occurred. According to Dr. Narang, this interpretation is supported by *Kumho Tire v. Carmichael*,[293] which according to Dr. Narang "tethered" the admissibility standard of expert testimony to the standards of medical practice, including the SBS/AHT studies on which he relies. This analysis is, however, incomplete.

To begin, *Daubert* governs only the general *admissibility* of scientific or expert testimony about the causes of injury or death in SBS/AHT cases. Increasingly, the legal issues do not focus on admissibility but focus instead on the case-specific significance of the evidence once it is admitted. These issues include whether medical opinions based on disputed medical issues are legally or factually sufficient to support convictions under the "beyond a reasonable doubt" standard[294] and whether previously obtained convictions should be re-examined given the new scientific understanding of the limitations of the triad as a diagnostic tool and the very real possibility of alternative explanations for a child's injuries or death. As a legal matter, in *Cavazos v. Smith,* six of the nine Supreme Court justices acknowledged flaws in

---

[291] *Daubert*, *supra* note 2.
[292] *Id.*
[293] Kumho Tire Co. v. Carmichael, 526 U.S. 137 (1999).
[294] *See* Tuerkheimer, *supra* note 37.

the evidence but held that the disputed SBS science presented at trial met the minimal due process standards for sufficiency of the evidence, at least as of the trial date.[295] Today, given the many challenges to the old SBS theory, the factual sufficiency of the evidence has become an increasingly significant question, as has the question of how to handle old convictions – a question not addressed by the majority in *Smith* beyond the narrow holding that the old expert opinions constituted sufficient evidence to convict as of the trial date and the suggestion that Ms. Smith seek clemency, which has since been granted. Given the changes in the science, old SBS/AHT convictions are now being challenged based on newly discovered evidence, actual innocence, ineffective assistance of counsel and other similar claims.[296]

In arguing admissibility under *Daubert*, moreover, it is unclear what Dr. Narang believes should be admitted. Evidence that some brain injuries in children are of traumatic origin, sometimes even intentionally inflicted? Evidence that subdural hematomas and retinal hemorrhages are seen in cases of inflicted abuse? Evidence that shaking can cause the triad and can lead to injury or death? Evidence that subdural hematomas and retinal hemorrhages are diagnostic of shaking or abuse in the absence of a major motor vehicle accident, fall from a multistory building or other proven alternative? Some of these questions are not controversial, and the evidence clearly satisfies the *Daubert* standard. Others are undermined by the research.

Dr. Narang's analysis of admissibility under *Daubert* further attempts to assess admissibility without limiting the evidence to be introduced or the purpose for which it is proffered. Under *Daubert,* however, any determination of admissibility must include an assessment of the significance of the evidence as it applies "to the task at hand."[297]

---

[295] *Smith* did not address the quality of the science, and admissibility was not an issue. Instead, the Court merely purported to apply, in a very straightforward manner, the deferential and forgiving constitutional standard for assessing sufficiency of the evidence under *Jackson v. Virginia.* Cavazos v. Smith, *supra* note 20, at 6. Under that standard, evidence will be deemed sufficient if, taking the evidence in the light most favorable to the prosecution, a reasonable jury could have found guilt beyond a reasonable doubt. Because the State offered experts who opined that the child died of SBS, the Court held that the jury could have found guilt if it credited those expert opinions, which the jury was free to do. The three dissenters – Justices Ginsburg, Sotomayor and Breyer – disagreed, suggesting that the changes in the literature and the fact-intensive character of the case called for a full briefing and consideration of the issues. Cavazos v. Smith, dissent, *supra* note 20, at 8, 9.

[296] State v. Edmunds, 746 N.W. 2d 590, 596 ¶ 15 (2008) (granting a new trial based on newly discovered evidence because "a significant and legitimate debate in the medical community has developed in the past ten years over whether infants can be fatally injured through shaking alone, whether an infant may suffer head trauma and yet experience a significant lucid interval prior to death, and whether other causes may mimic the symptoms traditionally viewed as indicating shaken baby or shaken impact syndrome"); State v. Louis, 332 Wis.2d 803 (Wis. Court of Appeals) (unpublished disposition).

[297] *See* Kumho, *supra* note 289 (quoting *Daubert, supra* note 2).

As Professor Michael Risinger explains, under *Daubert* and *Kumho*, "reliability cannot be judged globally, 'as drafted,' but only specifically, 'as applied.' *The emphasis on the judgment of reliability as it applies to the individual case, to the 'task at hand,' runs through the opinion like a river.*"[298] Because Dr. Narang's global analysis does not identify the specific propositions he wishes introduced or their application to the "task at hand," it tells us little about the admissibility of particular evidence in particular cases.

In determining these issues, clinical judgment cannot trump scientific research. To the contrary, under *Daubert,* the role of judgment or experience is limited:

> When a witness is called to ... make conclusions or inferences about adjudicative facts in the case at hand, the testimony is based in part on experience, but in part on some translation scheme to mediate between previous experiences and a particular conclusion in this case. In those circumstances, reliability is dependent on both sufficient experience and a reliable translation system. Perhaps where there are real-world, practice-based, empirically unambiguous indices of success or failure in coming to one's conclusions, we might rationally rely upon experience not only to provide the expert's data base, but also to authenticate the reliability of the conclusory skills involved. . . .
>
> *[But], in circumstances when experience alone does not resolve the main doubts about reliability, it would be irrational, and therefore an abuse of discretion to rely upon it.*[299]

It is also insufficient to rely on the fact that some professional groups accept or endorse the diagnosis of SBS/AHT. As Professor Risinger points out:

> [A]dherence to such standards cannot establish reliability [for admissibility purposes] when, as is often the case, it is the very reliability of the standard practice that is in issue. The guild test does at least claim to deal with reliability of the process beyond individual experience, but the reliability judgment is delegated to a group that, by definition, already believes in the process. The guild

[298] D. Michael Risinger, *Defining the "Task at Hand": Non-Science Forensic Science after* Kumho Tire Co. v. Carmichael, 57 WASH. & LEE L. REV. 767, 773 (2000) (footnote omitted; emphasis added).
[299] *Id.* at 775-76 (emphasis added).

> test trades the ipse dixit of the individual for the ipse dixit of the group.[300]

For this reason, *Kumho Tire* recognizes the inadequacy of general acceptance by a community when the issue is the reliability of the discipline and/or its application to the case at hand.[301]

In this response we do not take a position on the appropriate application of *Daubert* or other legal standards to particular hypotheses. We note, however, that there are essentially two possibilities. One could exclude both sides of the debate from the courtroom because there is inadequate information to make a conclusive diagnosis. Or, as is presently the case, experts with differing perspectives can argue it out in the courtroom, leaving it to judges and juries to sort out the intricacies of the infant brain and the complexities of biomechanics, as advocated by some prominent legal scholars, including Professor Edward Imwinkelried.[302] This approach presents two problems. First, trying and retrying undecided scientific issues on a weekly basis is extraordinarily expensive and inevitably results in inconsistent and "fluky" justice.[303] Second, and perhaps more important, if doctors cannot agree on these complex and unresolved issues, it is unlikely that jurors or judges can do any better.

What cannot be allowed is for supporters of the SBS/AHT hypothesis to present their hypotheses in the courtroom without making clear the limits of their knowledge and without the provision of competing presentations that are equally well-grounded and are often more consistent with the anatomy and physiology of the infant brain. Given the deference that judges and juries often give to expert opinion – a topic that is well-covered by Dr. Narang – the failure to present evidence from critics of the SBS/AHT hypothesis would almost certainly increase the number of false convictions in an area that is likely already riddled with false convictions.[304]

---

[300] *Id.* at 777.

[301] *Id.* at 778.

[302] *See* Imwinkelried, *supra* note 36.

[303] Tuerkheimer, *supra* note 95, at 523.

[304] While Dr. Narang dismisses the Goudge Inquiry in Ontario, Canada as consisting of "a few recent case reports of wrongful convictions" (Narang, *supra* note 3, at 515), the inquiry identified significant shortcomings in the field of pediatric forensic pathology and the diagnosis of shaken baby syndrome in particular. *See* Inquiry into Pediatric Forensic Pathology in Ontario (Sept. 2008) at http://www.attorneygeneral.jus.gov.on.ca/inquiries/goudge/index.html. The final report recommended a review of shaken baby and pediatric head injury convictions given the changes in SBS knowledge over the past two decades. *See* Consolidated Recommendations, Inquiry into Pediatric Forensic Pathology in Ontario 86 at *http://www.attorneygeneral.jus.gov.on.ca/inquiries/goudge/report/v1_en_pdf/Vol_1_Eng_CR.pdf.* Given the composition of the reviewing panel, it is unclear whether this review will lead to meaningful reform.

C. The Costs of Misdiagnosis.

The costs of misdiagnosing child abuse are obvious. If we under-diagnose child abuse, abusive parents will go unpunished and children will be left in unsafe homes. If we over-diagnose abuse, we destroy families and imprison innocent parents and caretakers. But there is a third often under-recognized cost of misdiagnosis: if we identify the wrong problem, we will inevitably apply the wrong solution. For example, when infection or stroke is misdiagnosed as abuse, the focus almost inevitably shifts from appropriate treatment to interrogations and arrests. If the misdiagnosis becomes systemic, this may be accompanied by a broader failure to identify medical problems that may ultimately prove to be preventable or treatable.

V. THE PATH FORWARD

As we work towards a new paradigm, we must bear in mind that the misdiagnosis of SBS/AHT is extraordinarily harmful, and that there is no self-corrective mechanism. Typically, any suggestion of SBS/AHT results in the automatic removal of the child and/or the child's siblings from the home. In addition to the emotional anguish, families often lose their savings and homes in frantic attempts to reclaim their children while facing prison sentences up to and including the death penalty. While these costs may be justified if a child has been abused or murdered, one should be quite certain that the abuse did indeed occur before imposing these costs, particularly given a legal system that is ill-equipped to correct past mistakes.[305]

In this case, the suggestion that shaking may harm vulnerable infants – a suggestion originally made by Dr. Guthkelch – was eminently sensible and holds true today. The SBS corollary – that shaking can be presumed from specific medical findings, including subdural hemorrhage – was plausible and widely accepted, including by Dr. Barnes and Dr. Squier, two of the co-authors of this article. Research conducted over the past decades has, however, established that the SBS hypothesis was based on a misunderstanding of biomechanics and the infant brain, and that there are many alternative causes. The shift in terminology from SBS to AHT has not solved this problem since it is *harder* – not easier – to defend against mechanisms that are not specified and that therefore cannot be tested or even debated.

---

[305] *See, e.g.*, Tuerkheimer, *supra* note 95, at 544 ("While not always expressly articulated, commitment to the finality of criminal convictions is deeply embedded in our criminal law structures and jurisprudence"); Cavazos v. Smith, *supra* note 20, at 7 (upholding conviction in Shirley Smith case despite acknowledging that "[d]oubts about whether Smith is in fact guilty are understandable").

We suggest four paths forward: research, collaboration, acknowledgment of the complexities, and learning to work under conditions of uncertainty.

A. Research

While we may never reach the levels of certainty demanded by evidence-based medicine or *Daubert*, we can certainly do better than we have done in the past. The research that Professor Goldsmith suggested in his NIH presentation in 2001 is as applicable today as it was then, and many of his suggestions align with those of Dr. Narang. Promising avenues include:

1. Studies on the anatomy and physiology of the infant brain, including the tolerance and failure limits of bridging veins, the role of cerebral spinal fluid, the mechanisms of retinal hemorrhage, and the role of biochemical cascades.
2. Analysis of other diseases and medical conditions that "mimic" SBS/AHT. While children are not little adults, they are subject to many of the same illnesses and medical conditions, including stroke, infection and nutritional deficiencies. We need to prevent, diagnose and treat these conditions rather than automatically ascribing them to abuse.
3. Careful, complete and nonjudgmental interviews of parents and caretakers, who often hold the clues to the correct diagnosis.
4. The development of protocols for investigating known alternative causes and identifying new causes.
5. Maintenance of a national registry on SBS/AHT cases, with retention of medical records, radiology images, blood samples and tissue samples. Videotaped autopsies would also be helpful. This would allow us to obtain accurate numbers and would provide a basis for ongoing evidence-based medical scrutiny and judicial review.

B. Working Together

To date, the child abuse community has been divided into hostile camps. If the medical issues are to be addressed, however, we need to work together. To do this, we endorse Dr. Guthkelch's recommendation that we adopt descriptive medical terminology that does not attempt to answer the question that is being asked. It is very difficult to have professional discussions on the cause of medical findings that are named "shaken baby syndrome" or "abusive head trauma" since these terms assume the causation.

Second, we need to continue to have less antagonistic professional discussions. The biannual conferences conducted by Penn

State Hershey are a good start: at these conferences, the organizers invite one or more presenters with diametrically opposed viewpoints to debate important issues. Often, the opposing camps are not as far apart as one might think. At the joint conference in Jackson Hole in 2009, for example, Dr. Plunkett and Dr. Dias quickly reached agreement that short falls can indeed be fatal, albeit rarely.[306]

Another constructive conversation occurred at a conference sponsored by the Queens District Attorney's Office in New York in September 2011. While the presenters and audience consisted largely of supporters of the SBS/AHT hypothesis, a panel composed of representatives from both sides of the debate discussed the key issues in a professional manner, sometimes reaching the same conclusions. For example, all of the panelists agreed that violent shaking may cause serious injury or death; that the triad is not diagnostic of abuse; and that each case requires an extended inquiry into the child's medical history and findings.

Third, personal and professional attacks on those with opposing views must stop. New ideas and a willingness to question traditional understandings are a precondition to scientific progress. If we are to ensure the wellbeing of children and families, our commitment to "getting it right" requires that we put aside our preconceptions and consider new ideas, including those contrary to our most cherished beliefs. While there is always resistance to new ideas, every mistake – and every delay in correcting our mistakes – imposes heavy costs on children and families. Debate and disagreement are essential, but there is no room for ad hominem attacks or efforts to prevent the dissemination of new research.

Finally, this debate needs to be taken to the broader legal, medical and scientific communities. Since we now know that our initial understanding of SBS/AHT was flawed, we need the advice and support of other specialties, including scientists and doctors who are not so closely involved in the debate. An independent review of the validity and basis for the SBS/AHT diagnosis by the National Academy of Sciences would be a good start. Discussions at major Children's Hospitals and other teaching hospitals would also be useful. In the legal arena, it is important to keep lawyers and the judiciary abreast of the advancing medical science and for prosecutors, judges and child protection agencies to consider the facts of each case rather than relying exclusively on medical hypotheses.

C. Acknowledging the Complexities

For decades, the SBS hypothesis provided a clear and simple explanation for the collapse or death of children who presented with

[306] Plunkett, *supra* note 111.

subdural hemorrhage, retinal hemorrhage and brain swelling. We now know, however, that its premises were wrong. The SBS hypothesis was based on a three-component model that did not reflect or recognize the complexities of the infant brain. In its original form, SBS taught that subdural hemorrhages were caused by the traumatic rupture of bridging veins in the "subdural space." However, the small thin subdurals typically found in infants are too small to represent the rupture of bridging veins, there is no subdural space between the dural and arachnoid membranes, and the "sub"dural hemorrhages in infants more likely originate in the venous dural plexus. The SBS hypothesis also taught that retinal hemorrhages in children were caused by the traumatic rupture of retinal veins. However, retinal hemorrhages in children are also seen in natural diseases and appear to reflect the same causes as retinal hemorrhages in adults, including lack of oxygen, thrombosis, increased intracranial pressure and time spent on life support. Finally, the SBS hypothesis taught that brain swelling was caused by the traumatic rupture of axons (nerve fibers) throughout the brain. However, we have known for more than a decade that the brain swelling is due to lack of oxygenated blood from any cause. All of this knowledge was neglected because it did not fit the model.

As our analyses become more anatomically correct, we are finding that there is no single model. Instead, the cases vary widely. A few cases present with large space-occupying subdural hemorrhages, as one would expect from ruptured bridging veins, but most present with thin intradural/subdural hemorrhages or thrombosed (clotted) veins with surrounding leakage. The ocular findings range from small unilateral retinal hemorrhages to bilateral multilayered retinal hemorrhages with retinochisis. The brain findings range from no brain damage at all to swollen hypoxic-ischemic brains with no hope of recovery. In some cases, all of the findings are acute (new) while in others, some or most of the findings are weeks to months old, or even older. The clinical histories are equally diverse: some children were healthy until their collapse; others had seizures, feeding difficulties or neurological impairments from birth; and yet others were symptomatic for days or weeks before collapse. In some cases, the collapse occurred when the child and a caretaker were alone; in others, the child and the caretaker were alone for minutes, if at all.

Given the heterogeneity of the medical findings and factual settings, one should be skeptical of a "one size fits all" diagnosis. One should also be skeptical of diagnoses that rest on three isolated findings without considering the characteristics of the developing brain and the relationship between the brain and the rest of the body. In so doing, one should remember that:

> If one were to name the universal factor in all death, whether cellular or planetary, it would certainly be loss of

> oxygen. Dr. Milton Helpern, who was for twenty years the Chief Medical Examiner of New York City, is said to have stated it quite clearly in a single sentence: "Death may be due to a wide variety of diseases and disorders, but in every case the underlying physiological cause is a breakdown in the body's oxygen cycle." Simplistic though it may sound to a sophisticated biochemist, this pronouncement is all-encompassing.[307]

In infant deaths, like all other deaths, the medical question is "what caused the lack of oxygen?" – not "who did it?" In our effort to determine why the child lacked oxygen – a question that has hundreds of possible answers and may sometimes prove unanswerable – we must treat each case the same way as we treat any other complex diagnosis: we must consider the lab results, the history, and all of the medical findings, bearing in mind the complexities of the human body and the physiological cascades that occur when this tightly regulated system goes awry. We must also carefully sort out, to the best of our ability, which findings help determine the cause of injury or death and which are secondary to an ongoing process and/or medical intervention. To do anything less is a disservice to children, families and our system of justice.

Today, everyone agrees that the "triad" of findings previously attributed to shaking may reflect abuse, accident or natural causes. What we don't know is how many cases – or sometimes which cases – fall into each of these categories. More than a decade ago, the Five Percenters suggested that 5% of SBS cases were misdiagnosed as child abuse[308] -- a figure that many thought was high. Based on the changes in the literature over the past decade, however, this figure may be even higher. But is it 10%, 25%, 50% or even 95%? The answer to this question is: we don't know. And until we do know, we cannot use statistics to address the issues, let alone to diagnose individual cases.

D. Working Under Conditions of Uncertainty

While we would all like a "gold standard" that distinguishes quickly and accurately between abuse, accident and natural causes, the medicine is uncertain and evolving, and the cases are complex. As we continue to search for answers, we need to make the best possible

---

[307] SHERWIN B. NULAND, HOW WE DIE: REFLECTIONS ON LIFE'S FINAL CHAPTER 67 (1994). Professor Nuland teaches surgery and the history of medicine at Yale University.

[308] Beth Hale, *Falsely Branded a Baby Batterer – Now Rioch Edwards-Brown's a Fighter for Justice,* DAILY MAIL, Nov. 24, 2011, at http://www.dailymail.co.uk/femail/article-2065430/INSPIRATIONAL-WOMEN-OF-THE-YEAR-Falsely-branded-baby-batterer--Rioch-Edwards-Browns-fighter-justice.html.

decisions under conditions of uncertainty. Dr. Narang suggests that we do this by emphasizing clinical judgment, leaving the resolution of the disputed medical issues to judges and juries. We suggest that the costs of this approach are too high and that we instead need to make clear the limits of our knowledge while expanding our knowledge base. In essence, this is what doctors and lawyers do when we treat patients or advise clients. It should be no different in the courtroom, where the safety of children and the futures of entire families hang in the balance.

EXHIBIT L

Infantile Subdural Haematoma and its Relationship to Whiplash Injuries

A. N. GUTHKELCH

British Medical Journal, 1971, 2, 430–431

Summary

Subdural haematoma is one of the commonest features of the battered child syndrome, yet by no means all the patients so affected have external marks of injury on the head. This suggests that in some cases repeated acceleration/deceleration rather than direct violence is the cause of the haemorrhage, the infant having been shaken rather than struck by its parent. Such an hypothesis might also explain the remarkable frequency of the finding of subdural haemorrhage in battered children as compared with its incidence in head injuries of other origin, and the fact that it is so often bilateral.

Introduction

Subdural haematoma is a commoner complication of head injury in infancy than at any other age. Lewin (1966) reported 47 cases of subdural haemorrhage in an unselected series of 1,750 head injuries occurring in patients of all ages—an incidence of about 2·7%—but Hendrick *et al.* (1964) found 5·2% in a large series of head-injured children ranging in age from birth to 15 years, the excess over Lewin's series being entirely accounted for by a greatly increased frequency in infants aged under 2 years, and particularly in those under 6 months, in which latter group it was as high as 20%. But there is no report of an incidence of subdural haematoma complicating road traffic accidents, etc., in very young children which approaches the 42% quoted by Weston (1968) in his series of necropsies of fatal cases of child assault, which is a particularly remarkable figure when one considers that the children in Weston's series ranged in age from 2 months to 5 years, with an average of 24 months—that is, considerably older than the high-risk group of Hendrick *et al.*—and that in some of them a head injury did not seem to have been noticed before death.

The commonest cause of infantile subdural haemorrhage is rupture of one or more of the delicate bridging veins which run from the cerebral cortex to the venous sinuses, the mode of injury being either a single acceleration or deceleration due to a heavy moving object striking the head or the rapidly moving head being brought up against a stationary mass; multiple applications of force would increase the total strain on the bridging veins and might result in an increased incidence of rupture. But direct violence is not an essential part of the picture. It has been known for some time that chronic subdural haematomas commonly occur in adults after disproportionately slight head injuries, sometimes when there is no reason to suppose that the head was ever injured at all. Meredith (1951) reported one such case in an adult after a fall on to the buttocks, and a senior American neurosurgeon (German *et al.*, 1964) developed a subdural haematoma after his head had been jerked by the violent motion of the "bobsled" which he was riding at a fun fair.

Combined Neurological Service, Hull Royal Infirmary, Hull
A. N. GUTHKELCH, M.CH., F.R.C.S., Consultant Neurosurgeon

More recently Ommaya and Yarnell (1969) published two well-documented cases of subdural haematoma, in both of which the subject sustained a whiplash injury to the neck as a result of an automobile accident, the head itself not being injured at all. There was no immediate loss of consciousness and it was not until several days later that cerebral signs developed.

It is now submitted that the conditions which are known to exist in many cases of the battered child syndrome are particularly favourable to the production of subdural haematoma in infants by an essentially similar mechanism. Kempe *et al.* (1962) noted that in their experience the extremities of the child are often used as handles during an episode of rough treatment; "sometimes an arm is pulled to jerk a reluctant child to his feet, sometimes the legs are held while the tiny body is swung in a punitive way." In one of Caffey's (1946) cases of subdural haematoma the history is given in the following words: "Baby rolled off a table and his mother grabbed him by the forearm and jerked him in the air to prevent his fall." It seems clear that the relatively large head and puny neck muscles of the infant must render it particularly vulnerable to whiplash injury in this sort of situation. Moreover, since one would expect that the child is often grasped more or less symmetrically by chest or limbs the rotation-acceleration strains on the brain would tend to occur fairly symmetrically also, in an anteoposterior direction. This may be the reason why infantile subdural haematoma is even more often bilateral—for example, in 14 out of 18 cases (78%) in my earlier series (Guthkelch, 1953)—than subdural haematoma in adults, for which the proportion of bilateral cases does not exceed 50%.

One cannot say how commonly assault in the form of violent shaking rather than of direct blows on the head is the cause of subdural haematoma in infants who are maltreated by their parents. Possibly it will be found that the frequency of this mechanism varies between different nations according to their ideas of what is permissible, or at least excusable, in the treatment of children. Among the children mentioned in Weston's (1968) detailed records there were three—a 4-month-old girl, a 5-month-old boy, and a 2-year-old girl—in whom the cause of death was subdural haemorrhage, it being admitted by the assailant that the child had been violently shaken. In only one of these was there any mark of injury on the head, and this is described as a superficial bruise of the scalp, the baby's head having apparently struck the side of her cot, as it were, incidentally. Court (1969) quoted a mother's confession that she was in the habit of shaking her babies "in an insane rage."

Patients

Twenty-three cases of proved or strongly suspected parental assault on children all under the age of 3 years, and all except one under 18 months, were admitted to the Hull Royal Infirmary between June 1967 and May 1970. Subdural bleeding had occurred in 13 (57%) of these and was bilateral in 10, the only sort of injury which was recorded more often being bruising of the skin. One or more fractures of long bones were found in association with subdural haematoma in six cases.

There were eight children in whom a fracture of the skull was discovered, and six of these had a subdural haematoma. Of the seven children suffering from subdural haemorrhage who had no skull fracture five had no external marks of injury

on the head either, though in one of these necropsy showed surface haemorrhages on the cerebral cortex, and another was found at operation to have extensive cortical bruising. Though 8 out of 16 battered babies with definite evidence of head trauma had a subdural haematoma, in 5 out of 13 cases of subdural haemorrhage no evidence of the application of direct violence to the head was forthcoming.

In two additional personal cases there was very strong reason indeed to suppose that the mechanism of production of the subdural haemorrhage had been by shaking rather than battering.

CASE 1

A 6-month-old boy was found by his father lying unconscious in his cot and having repeated convulsions. His mother was sitting beside him too shocked to speak or move. On admission to hospital he was found to be well nourished and well cared for, without any external marks of injury. He was stuporous and breathing heavily, with a temperature of 38°C. There was pronounced hyperreflexia of all limbs and a tense fontanelle. Immediate subdural taps showed almost solid haematomas on both sides, but despite the evacuation of these he died three days later.

At necropsy several cortical bridging veins were found to have been torn from their dural attachments near the falx cerebri and the surface of the underlying brain was contused, with some small lacerations. There were no other internal injuries of any sort though some mucopus was present in the upper respiratory passages. The mother eventually admitted that the baby had had several fits of coughing and said that she feared that he was going to choke. She therefore held him up and shook him several times in order to try to clear his throat, whereupon he went into convulsions.

In this case the possibility of compression of the thorax with a consequent rise in jugular venous pressure and rupture of the cortical veins is not excluded, but there were no signs of bruising of the chest wall or of the lungs, nor any rib fractures. It was felt that the mother's account was probably truthful and that the tragedy occurred because of her lack of realization of the damage that can result from rough handling of a small baby. It has indeed been repeatedly observed that the parents of battered children may handle them exceedingly clumsily, even when they are under observation during their visits to hospital.

CASE 2

A 6-month-old boy was admitted to hospital on account of vomiting and convulsions, the suspected diagnosis being one of meningitis. Again there were no external signs of injury and no fractures of the skull or long bones, but the combination of a tense bulging fontanelle and bilateral retinal haemorrhages gave rise to a suspicion of subdural haematoma, which was confirmed and duly treated. At this time no suspicion of parental violence was entertained, but soon after his discharge from hospital the patient's twin brother was admitted suffering from a fracture of the femur for which his parents could give no explanation, and shortly afterwards the patient himself was readmitted with recurrent subdural haematomas on both sides. This time there were oval bruises on each of his forearms which fitted the pads of the fingers and thumb of the examiner's hand when the limbs were gripped. The parent denied having struck or beaten him but eventually his mother admitted that she and her husband "might have" shaken him when he cried at night.

Comment

The mere absence of visible injury on the head does not exclude direct violence, for Weston (1968) pointed out that in fatal cases "examination of the galea frequently revealed numerous . . . haemorrhages . . . even in the absence of conspicuous external bruising, abrasion or laceration." In a non-fatal case there is, of course, no opportunity to expose and examine the deep layers of the scalp, particularly now that craniotomy has been almost completely abandoned in the treatment of infantile subdural haematoma. None the less, one has the impression that "a good shaking," is felt, at least by British parents, to be socially more acceptable and physically less dangerous than a blow on the head or elsewhere.

As applied to the complicated mechanics of the brain and its coverings, subjected to the many different forces which constitute the causes of head injury, a limited series of experiments with a simple model proves nothing. Nevertheless, the following device, for the idea of which I am indebted to Dr. A. K. Ommaya, who has used a similar one to demonstrate the value of head restraint in preventing cerebral commotion in automobile accidents, may be found instructive.

An ordinary round-bottomed litre glass flask is filled with liquid paraffin in which have been suspended a few spoonsful of desicated coconut. When this mixture has been agitated the flakes will remain stationary in a state of even dispersion for periods of several minutes at a time. The flask is completely filled, closed with a rubber bung, and held firmly by the neck. It will be found that the flakes can be more readily set in motion, and will continue to swirl about for longer, after shaking for a few seconds than after the hardest blow that can be delivered without breaking the flask.

The phenomena observed are essentially similar to the movements of the brain which have been shown in anaesthetized monkeys in which the scalp and the top of the calvarium had been replaced by a Perspex window and which were then subjected to sudden accelerations (Ommaya *et al.*, 1968).

Conclusion

It has been shown that there is a discrepancy between the frequency of subdural haematoma occurring in battered children and of the same condition complicating head injuries of other origin, the incidence in the former being unexpectedly high, though in most of those in whom there was no actual skull fracture there was not even clear evidence of the application of direct violence to the head. This suggests that when the head is not the main target of attack the likely mechanism of production of the haematoma is one in which repeated sheering strains of one sort or another are applied to the cranial contents.

It follows that since all cases of infantile subdural haematoma are best assumed to be traumatic unless proved otherwise it would be unwise to disregard the possibility that one of these has been caused by serious violence, repetition of which may prove fatal, simply on the basis that there are no gross fractures or other radiological bone changes in the limbs, nor any fractures of the skull. One must keep in mind the possibility of assault in considering any case of infantile subdural haematoma, even when there are only trivial bruises or indeed no marks of injury at all, and inquire, however guardedly or tactfully, whether perhaps the baby's head could have been shaken.

I wish to acknowledge the co-operation of Dr. M. G. Philpott and Dr. R. J. Pugh, who permitted me to quote the series of battered children admitted under their care to Hull Royal Infirmary, and also of Mr. W. Tuffnell and Mr. L. Hodge, of Hull University, who prepared the model.

References

Caffey, J. (1946). *American Journal of Roentgenology and Radium Therapy*, **56**, 163.
Court, J. (1969). *Medical Social Worker*, **22**, 15.
German, W. J., Flanigan, S., and Davey, L. M. (1964). *Clinical Neurosurgery*, **12**, 344.
Guthkelch, A. N. (1953). *British Medical Journal*, **1**, 233.
Hendrick, E. B., Harwood-Hash, D. C. F., and Hudson, A. R. (1964). *Clinical Neurosurgery*, **11**, 46.
Kempe, C. H., Silverman, F. M., Steele, B. F., Droegemueller, W., and Silver, H. K. (1962). *Journal of the American Medical Association*, **181**, 17.
Lewin, W. (1966). *The Management of Head Injuries*, p. 77. London, Baillière, Tindall, & Cassell.
Meredith, J. M. (1951). *Journal of Neurosurgery*, **8**, 444.
Ommaya, A. K., Faas, F., and Yarnell, P. (1968). *Journal of the American Medical Association*, **204**, 285.
Ommaya, A. K., and Yarnell, P. (1969). *Lancet*, **2**, 237.
Weston, J. T. (1968). In *The Battered Child*, ed. R. E. Helfer and C. H. Kempe, p. 77 and Appendix B, p. 227. Chicago, University of Chicago Press.

EXHIBIT M

"Shaken Baby Syndrome": Do Confessions by Alleged Perpetrators Validate the Concept?

Jan E. Leestma, M.D., M.M.

ABSTRACT

The concept that a certain constellation of findings develops immediately after a baby is shaken, with no impact to the head, is based solely on confessions or admissions by alleged perpetrators. The reliability of confessions in the setting of interrogation by law-enforcement officials is questionable. A review of the literature reveals very few cases of admissions of "pure" shaking. Methodologic flaws preclude any definitive conclusions about causation from these cases.

The Origins of the "Shaken Baby Syndrome"

The "shaken baby syndrome" (SBS) is embedded in the collective minds of the public, law enforcement, prosecuting attorneys, child-protection personnel, and physicians.

The concept was largely derived from the papers of Caffey,[1-5] a radiologist specializing in pediatric cases. He had for years suggested that the multiple limb fractures he observed in babies were the result of abuse. It was not until Kempe and his associates at the University of Colorado Medical Center in Denver published their seminal article in 1962[6] that the theories of Caffey and others began to gain acceptance.

A sensational case of a child-care nurse who admitted to shaking and killing three babies in her care attracted Caffey's[5] attention, and he apparently thought that her admission explained what he had been seeing for years but had been unable to explain. Caffey now theorized that multiple fractures of the lower extremities and other bones as well as subdural hemorrhages in apparently abused babies were caused by the flailing legs and a head flopping back and forth with shaking. This mechanism, without any scientific evidence to support it, made intuitive sense to him and others,[7,8] and thus was born "whiplash-shaking" and later the "shaken baby" syndrome.

SBS has been embraced by the American Academy of Pediatrics[9] and other organizations. They support the concept that manual shaking of a baby causes a "constellation" of rapidly evolving processes (subdural hemorrhages, retinal hemorrhages, brain swelling, metaphyseal long-bone fractures, respiratory failure, and often death) in the apparent absence of signs of external injury, and that these things collectively cannot occur in any other context than abuse.

Conservatively, more than 100 case reports and small series in support of Caffey's theories have appeared in the literature from the early 1970s to 2001. A number of them provided perpetrator admissions of shaking that seemed to validate the concept of shaking as a cause of the syndrome. However, a literature that challenges the mechanism of injury, the components of SBS, and even the existence of the syndrome itself has also developed. These challenges are in the form of biomechanical analyses,[10-14] criticism of case-analysis methodology,[15] analysis of admissions of alleged perpetrators,[16] and a consideration of alternate explanations.[10-11] It is safe to say that there is a deep divide between proponents of the SBS and its critics.

A Review of the "Confessional" Literature

Several recent studies have attempted to correlate "confessions" of perpetrators with injuries typically found in cases deemed to be SBS. These studies have been regarded by many as strong support for the theory of SBS.[22-23] Of prime concern in these papers, however, is the basic issue of the value of a supposed confession in determining a mechanism for injury.

With any confession or admission, there is the issue of veracity. Accused individuals are well known to fabricate historical information, augment certain aspects of what they might have done, say what they think an interrogator wants to hear, or omit important facts, presumably to give a better impression than might otherwise be the case.[24,25]

Furthermore, though it is difficult to fathom why, accused individuals may confess to things they didn't do. The issue of duress in its many forms in such cases is a valid one.[26] One scenario in alleged SBS cases is that an interrogator (policeman, child-abuse professional, or child-protection professional) may employ subterfuge to secure an admission of shaking. Deceit is not uncommon, as when the interrogator may communicate to the accused that "if you could tell us exactly what happened and if you shook the baby, we could do something for the baby and maybe save its life." There are, of course, no specific treatments in such cases other than those already being given to the baby, and this type of suggestion is disingenuous at best.

There may be other instances in which the results of interrogations and supposed statements by an accused are not what the accused said at all. Thus it is virtually impossible to determine what in an admission or confession is true and complete, false, fabricated, or tainted. To base an injury causation study on such information does not meet the accepted standards of analysis or interpretation because of insoluble issues of bias. This does not say that admissions/confessions are useless, only that validity and helpfulness depend on the context and the use being made of the information.

In a recent report of Biron and Shelton,[22] the authors concluded that shaking alone can produce serious neurological impairment or death. They based their study on 52 cases collected in Queensland, Australia, over a 10-year period (1993-2003). The population analyzed was defined as those children under the age of 2 years who were judged to be homicides or assaults. A team of child-protection personnel, pediatricians, welfare, and law-enforcement professionals evaluated the cases. Transcripts and tape-recorded interviews with witnesses and perpetrators were reviewed, as were autopsy reports when they were available.

The authors classified those babies as having been injured by "shake-only" by the presence of subdural and/or subarachnoid hemorrhage, retinal hemorrhages, and absence of medical (skull or scalp) injury or witness evidence of impact. The remaining cases were classified as "impact only," as determined by skull or scalp injury, perpetrator or witness evidence of an impact without associated shaking, and the absence of retinal hemorrhages. "Shake-impact" cases were identified if they had combinations of the above. Cases with insufficient evidence were called "indeterminate." Twenty of 52 babies died.

The authors concluded that of the 52 cases, 13 were "shaken-only" (five deaths), 3 had head "impact only" (one death), and 25 had evidence of both "shaking and impact" (ten deaths), with 11 cases judged "indeterminate" as to cause (four deaths). In five of the 13 "shaken-only" cases recorded, perpetrator confessions were obtained; these five cases and two more were presented in detail.

The authors cited a number of papers that have questioned, from various perspectives including biomechanics, the validity of the concept of SBS but appear not to have been sufficiently impressed to take these criticisms to heart before making their conclusions. The authors did not cite an important paper by Donohoe,[15] which discussed in detail methodological issues in studies like theirs and basically concluded that the existing literature on SBS does not meet sufficiently rigorous standards to conclude that shaking alone causes intracranial injury.

The Biron and Shelton paper[22] has many serious flaws that include selection bias, observer bias, lack of controls, failure to evaluate causal possibilities beyond shaking, and circular reasoning, to name a few. The assertion that retinal hemorrhages are a discriminator for shaking has been challenged in the literature for years, as have other selection criteria.[13,14,16,21]

The paper is little different from most of the literature based on case series that try to support the concepts of SBS in that the principles and methods of science were not adhered to and the data presented do not justify the conclusions reached.[27,28] These same problems apply to most of the literature that makes use of confessions as a justification that shaking alone causes intracranial injuries.[23]

Another recent case analysis,[16] encompassing more than 30 years of published case reports of presumably abused babies, approached the admissions issue from another perspective. Plunkett found 54 instances, in 324 cases with individual case data, of an admission by someone that he had shaken the injured baby in some fashion. In the 270 remaining cases, no record of any admission was reported.

The study found that the reported information in admissions varied widely in the amount of precise information provided and in the context of the shaking: for example, attempts to revive, shaking after a violent event such as strangulation/smothering or throwing the baby to the floor had occurred, and incidental shaking or bouncing during play. It was found that in 11 cases shaking had apparently occurred without evidence of impact. In 12 cases, only shaking was admitted, but head impact was found nonetheless. In 18 cases, admissions of shaking were documented, but there was no information, pro or con, about head impact injury, so these cases could not be analyzed.

An additional 13 cases, those of Hadley et al.,[29] in which admissions of shaking may have occurred, were not included in the analysis because of ambiguous statements in the article regarding what constituted an admission of shaking, or even whether it occurred at all.

It is significant that in the 11 shaken and possibly not head-impacted babies, all but three survived, thus one cannot be sure that no head impact had occurred in the eight survivors. In the three who died, apparently none had head impacts at autopsy. Thus, these may be the only cases that might qualify as "pure" and might have sustained their injuries by shaking alone. It should be obvious that with a case population this small, few robust conclusions about causality can be reached.

Time of Onset of Symptoms and Signs

A common theme that emerges from the "confessional" literature[22,23] is the alleged immediacy of the appearance of symptoms after a reported shaking episode as reported by the perpetrator. A common allegation of some child-abuse experts is that all or virtually all shaken babies become ill immediately after having been shaken.[9,30] Therefore, the individual present when the child decompensates is responsible. The published case literature does not support this contention.

In the Leestma study,[16] of the 11 babies who might qualify as "pure" shaken babies only four had information about a possible interval between shaking and the appearance of symptoms. None of these babies showed immediate symptoms and were reported to have developed symptoms a day or more after shaking. Considering all 54 admitted shaken babies, only 12 case reports gave information about the time of onset of the symptoms. Only two cases showed immediate symptoms, and all the rest showed delays from hours to days or longer after the supposed shaking episode. One should bear in mind that most of these babies had impact injuries to the head. Even in this circumstance, where an obvious head impact occurred, symptoms did not always appear immediately. Others have also reported these observations.[31,32]

Differential Diagnosis

It should be apparent that from virtually every perspective many flaws exist in the theory that shaking is causative. No case studies have ever been undertaken to probe even a partial list of

possible confounding variables/phenomena, such as the presence of intracranial cysts or fluid collections, hydrocephalus, congenital and inherited diseases, infection, coagulation disorders and venous thrombosis, recent immunizations, medications, birth-related brain injuries, or recent or remote head trauma. Until and unless these and probably many more factors are evaluated, it is inappropriate to select one mechanism only and ignore the rest of the potential causes.

Conclusions

The confessions or admissions of a perpetrator are at best tenuous support for the shaking mechanism for infantile head injury. A critical appraisal of any literature that proposes a causal mechanism of shaking for brain injury must include an evaluation of case selection methodology, population or sample size, possible case control issues, data analysis methods, and whether the conclusions reached are justified by the data presented.[2 8]

Another vital issue, often overlooked, is a critical evaluation of literature cited. Unless the reader is very well informed on the issues and is intimately familiar with the literature, this component of an informed appraisal of an article almost never gets done, and the reader may accept the conclusions uncritically.

Jan E. Leestma, M.D., M.M., is board certified in anatomic and neuropathology, and is a consultant in neuropathology at Children's Memorial Hospital of Northwestern University Medical Center, Chicago.

Potential Conflict of Interest: Dr. Leestma has been engaged to provide expert testimony in various criminal and civil cases, including some involving alleged SBS. Contact: jleestma@aol.com.

REFERENCES

1. Caffey J. Multiple fractures in the long bones of infants suffering from chronic subdural hematoma. *Am J Radiol* 1946;56:163-173.
2. Caffey J. Some traumatic lesions in growing bones other than fractures and dislocations: clinical and radiological features. *Br J Radiol* 1957;30:225-238.
3. Caffey J. On the theory and practice of shaking infants: its potential residual effects of permanent brain damage and mental retardation. *Am J Dis Child* 1972;124:161-169.
4. Caffey J. The parent-infant traumatic stress syndrome (Caffey-Kempe syndrome) (battered baby syndrome). *Am J Radiol* 1972;114:218-229.
5. Caffey J. The whiplash shaken infant syndrome: manual shaking by the extremities with whiplash-induced intracranial and intraocular bleedings, linked with residual permanent brain damage and mental retardation. *Pediatrics* 1974;54:396-403.
6. Kempe CH, Silverman FN, Steele BF, Droegemueller W, Silver HK. The battered child syndrome. *JAMA* 1962;181:105-112.
7. Guthkelch AN. Infantile subdural haematoma and its relationship to whiplash injuries. *BMJ* 1971;2:430-431.
8. Eisenbrey AB. Retinal hemorrhage in the battered child. *Child's Brain* 1979;5:40-44.
9. American Academy of Pediatrics (Committee on Child Abuse and Neglect: Kairys SW, Alexander RC, Block RW, et al.): Shaken baby syndrome: rotational cranial injuries—technical report (T0039). *Pediatrics* 2001;108:206-210.
10. Duhaime AC, Gennarelli TA, Thibault LE, et al. The shaken baby syndrome: a clinical, pathological, and biomechanical study. *J Neurosurg* 1987:66:409-415.
11. Prange MT, Coates B, Duhaime AC, Margulies SS. Anthropomorphic simulations of falls, shakes and inflicted impacts in infants. *J Neurosurg* 2003;99:143-150.
12. Bandak FA. Shaken baby syndrome: a biomechanics analysis of injury mechanisms. *Forensic Sci Int* 2005;151:71-79.
13. Ommaya AK, Goldsmith W, Thibault L. Biomechanics and neuropathology of adult and paediatric head injury. *Brit J Neurosurg* 2002;16:220-242.
14. Goldsmith W, Plunkett J. A biomechanical analysis of the causes of traumatic brain injury in infants and children. *Am J Forensic Med Pathol* 2004;25:89-100.
15. Donohoe M. Evidence-based medicine and shaken baby syndrome. Part I. Literature review 1966-1998. *Am J Forensic Med Pathol* 2003;29:239-242.
16. Leestma, JE. Case analysis of brain injured, admittedly shaken infants: 54 cases. *Am J Forensic Med Pathol* 2005;26:199-212.
17. Scheibner V. Shaken baby syndrome diagnosis on shaky ground. *J Austr Coll Nutr & Env Med* 2001;2:5-8,15.
18. Duhaime AC, Gennarelli TA, Sutton LM, Schut L. The "shaken baby syndrome": a misnomer? *J Pediatr Neurosciences (Riv. Neuroscienze Pediariche—It)* 1988;4:77-86.
19. Plunkett J. Fatal head injuries caused by short-distance falls. *Am J Forensic Med Pathol* 2001;22:1-12.
20. Tongue AC. Guest editorial: the ophthalmologist's role in diagnosing child abuse. *Ophthalmology* 1991;98:1009-1010.
21. David TJ. Shaken baby (shaken impact) syndrome: non-accidental head injury in infancy. *J Roy Soc Med* 1999;92:556-561.
22. Biron D, Shelton D. Perpetrator accounts in infant abusive head trauma brought about by a shaking event. *Child Abuse Negl* 2005;29:1347-1358.
23. Starling SP, Patel S, Burke BL, et al. Analysis of perpetrator admissions to inflicted traumatic brain injury in children. *Arch Pediatr* 2004;158:454-458.
24. Conti RP. The psychology of false confessions. *J Credibility Assessment Witness Psychol* 1999;2:14-36.
25. Kassin SM. On the psychology of confessions: does innocence put innocents at risk? *Am Psychol* 2005;60:215-228.
26. Leo RA, Ofshe RJ. The consequences of false confessions: deprivations of liberty and miscarriages of justice in the age of psychological interrogation. *J Crim Law & Criminol* 1998; 88:429-497.
27. Foster KR, Huber PW. *Judging Science. Scientific Knowledge and the Federal Courts.* Cambridge, Mass.: MIT Press; 1997.
28. Greenhalgh T. *How to Read a Paper: The Basics of Evidence-Based Medicine*. London, England: BMJ Publishing Group; 1997.
29. Hadley MN, Sonntag VKH, Rekate HL, Murphy A. The infant whiplash-shake injury syndrome: a clinical and pathological study. *Neurosurgery* 1989;24:536-540.
30. Lazoritz S, Palusci VJ, eds. *The Shaken Baby Syndrome. A Multidisciplinary Approach.* New York, N.Y.: Haworth Maltreatment & Trauma Press; 2001.
31. Howard MA, Bell BA, Uttley D. The pathophysiology of infant subdural haematomas. *Brit J Neurosurg* 1993;7:355-365.
32. Greenes DS, Schutzman SA. Occult intracranial injury in infants. *Ann Emerg Med* 1998;32:680-686.

EXHIBIT N

Ashdin Publishing
Journal of Forensic Biomechanics
Vol. 2 (2011), Article ID F110601, 9 pages
doi:10.4303/jfb/F110601

Research Article

Biomechanical Evaluation of Head Kinematics During Infant Shaking Versus Pediatric Activities of Daily Living

John Lloyd,[1] Edward N. Willey,[2] John G. Galaznik,[3] William E. Lee III,[4] and Susan E. Luttner[5]

[1]*32824 Michigan Avenue, San Antonio, FL 33576, USA*
[2]*6727 1st Avenue South, Suite 204, St Petersburg, FL 33707, USA*
[3]*Student Health Center, University of Alabama, Box 870360, Tuscaloosa, AL 35487-0360, USA*
[4]*Department of Chemical & Biomedical Engineering, University of South Florida, 4202 E. Fowler Avenue, Tampa, FL 33620, USA*
[5]*4035 Orme Street, Palo Alto, CA 94306, USA*
Address correspondence to John Lloyd, drjohnlloyd@tampabay.rr.com

Received 29 June 2011; Revised 14 November 2011; Accepted 15 November 2011

Abstract Abusive shaking of infants has been asserted as a primary cause of subdural bleeding, cerebral edema/brain swelling, and retinal hemorrhages. Manual shaking of biofidelic mannequins, however, has failed to generate the rotational accelerations believed necessary to cause these intracranial symptoms in the human infant. This study examines the apparent contradiction between the accepted model and reported biomechanical results. Researchers collected linear and angular motion data from an infant anthropomorphic test device during shaking and during various activities of daily life, as well as from a 7-month-old boy at play in a commercial jumping toy. Results were compared among the experimental conditions and against accepted injury thresholds. Rotational accelerations during shaking of a biofidelic mannequin were consistent with previous published studies and also statistically indistinguishable from the accelerations endured by a normal 7-month-old boy at play. The rotational accelerations during non-contact shaking appear to be tolerated by normal infants, even when repetitive.

Keywords biomechanics; traumatic brain injury; TBI; shaken baby syndrome; SBS; abusive head trauma; AHT; activities of daily living; ADL

1 Introduction

Shaken baby syndrome (SBS) has been defined as the presence of three specific findings: subdural hematoma (SDH), cerebral edema/brain swelling and retinal hemorrhage (RH) [8,25]. This injury cluster—sometimes referred to as "the triad"—has been presumed to occur as a result of abusive shaking but not as a result of household falls, even falls down stairs [7]. The shaking hypothesis, first proposed in the early 1970s [6,24], was seemingly accepted as settled science in 2001 in two documents: a position paper from the National Association of Medical Examiners [7] and an updated position statement from the American Academy of Pediatrics (AAP) [2]. Although the hypothesis has never been scientifically proven, practitioners working with the accepted model have accumulated years of clinical experience that convinces them that the model is correct [5].

Over the past 25 years, however, biomechanical research studies and computer modeling have raised questions about traditional thinking regarding SBS. When Duhaime et al. tried to confirm the shaking hypothesis in the 1980s using anthropomorphic test devices (ATDs), adult subjects failed to generate sufficient angular acceleration by shaking to reach the predicted thresholds for infant subdural hematoma (SDH) and diffuse axonal injury (DAI) [16]. A follow-up study published in 2003 concluded that non-contact shaking or a fall from less than 1.5 meters were less likely to cause injury than inflicted slamming against a hard surface [36].

Another team replicating Duhaime's work using alternative dummy designs, including different necks, recorded slightly higher accelerations in non-contact shaking, but peak accelerations were recorded when the surrogates' heads hit their own chests and backs [11]. Since head impact is known to cause SDH and DAI, researchers have encouraged the use of a more generic term for the symptom cluster, such as shaking-impact syndrome [15] or abusive head trauma (AHT) [9].

Computer modeling has explored refinements to the single-hinge ATD neck designs used in the Duhaime [16], Prange [36] and Cory [11] studies, predicting that more realistic neck designs, such as that used in the CRABI biofidelic mannequin, would yield lower angular accelerations [40]. As predicted, physical trials with a CRABI-6 yielded lower peak angular accelerations than what Duhaime reported (574.8 rad/s^2 [16] versus 1,138 rad/s^2 [10,28]). Peak values across trials with the Aprica 3.4 kg anthropomorphic test device (1,436.5 rad/s^2 [28]), however, exceeded Duhaime's peak magnitude slightly, and trials with the Aprica 2.5 kg

model (13, 252 rad/s^2 [28]) exceeded Duhaime's by an order of magnitude, although these figures seem to represent a different calculation strategy.

Biomechanical computer modeling has also concluded that a child's neck would break at forces lower than those required to produce the intracranial injuries associated with SBS [3,35]. Cadaveric studies have quantified the mechanical properties and strength of the human infant neck [17,31, 34], supporting the supposition that structural neck failure would result from even the accelerations reported during shaking of the Aprica 3.4 [28].

Direct experiments to determine brain injury thresholds in infants are prohibited for obvious ethical reasons. Data from field monitoring of adults and adolescents participating in contact sports have been used to set injury thresholds for mild traumatic brain injury in the more mature brain [18, 41]. Based on human data, not extrapolations from animal research, these thresholds for mild traumatic brain injury are 5–10 times greater than the rotational accelerations reported from abusive shaking of infant ATDs, particularly in studies that used a more realistic neck design. Nevertheless, the infant brain could be more vulnerable to injury from rotational accelerations than the adult and youth brain, and the repetitive accelerations presumed in shaking might not be comparable to sports contact.

Accident reconstructions of airbag-deployment injuries to infants have produced indirect data on infant injury thresholds. Klinich et al. concluded that infants in rear-facing car seats can tolerate up to 45 resultant Gs of acceleration without head injury, but may sustain fatal injury, including skull fracture and SDH, when 100 Gs or more results [14]. The auto industry's data, unfortunately, does not separate rotational from translational components. Further, the accident scenarios did not produce repetitive accelerations, and results did not reveal where the actual injury threshold might lie between 45 Gs and 100 Gs. This auto industry data is provocative, however, because it indicates that infants survive up to 45 resultant Gs, which is more than 4 times the 9.29 G and 9.9 G linear accelerations that are reported in the abusive shaking studies by Duhaime [16] and Jenny [28], respectively.

Depreitere et al. estimated the rupture threshold for adult bridging veins at 10,000 rad/s^2 [13], the same figure used by Duhaime et al. for concussion in infants [16]. Researchers have worked with even higher thresholds for infant SDH [11,16,40].

In the present study, researchers collected data from infant surrogates during abusive shaking simulations and various activities of daily living (ADLs) commonly experienced by infants. These ADLs would not be defined as abusive and would not be predicted to cause injury. Taking measurements during these activities establishes a baseline for commonly generated linear and angular head motions, allowing researchers to compare these values to injury thresholds, an approach also used by researchers trying to understand whiplash accelerations in automobile accidents [1].

	Height (in)	Weight (lb)
NCSBS demonstration doll	21.0	2.0
CRABI—12 month	29.5	22.0
KL (7-month-old baby boy)	27.0	19.2

Table 1: Anthropometry of infant surrogates.

Another data set was collected from an actual human infant at spontaneous play. He set his own level of activity and energy investment, absent any expressions of anxiety, discomfort, or neurological dysfunction. This infant's data were recorded, including rotational acceleration, which is usually considered to have the greatest potential to cause brain injury and SDH [27]. These rotational accelerations were specifically repetitive, addressing one of the criticisms of extrapolations from impact studies. Remarkably, this infant's spontaneous play resulted in rotational accelerations similar to those reported during the shaking of a 6-month CRABI biofidelic mannequin [28], and statistically undifferentiated from our measurements during shaking of a 12-month CRABI biofidelic mannequin. While our data does not establish a threshold for brain injury from repetitive rotational accelerations, it does establish a level of repetitive and cumulative rotational acceleration that is clearly well tolerated without apparent injury.

2 Materials and methods

2.1 Infant representatives

Researchers collected data from three infant representatives:

(i) a Child Restraint/Airbag-Interaction (CRABI)-12 biofidelic mannequin, weighing 22 pounds and measuring 29 inches head to toe, calibrated by and purchased from Denton ATD, Plymouth, MI, USA;

(ii) a demonstration doll, weighing 2 pounds and measuring 21 inches, sometimes allowed in court for demonstration purposes, purchased from the National Center on Shaken Baby Syndrome (NCSBS), Ogden, UT, USA;

(iii) a 7-month-old infant male, KL, weighing 19.2 pounds and measuring 27 inches, playing in his Fisher Price Deluxe Jumparoo (Mattel, Inc., El Segundo, CA, USA).

Table 1 lists the basic anthropometry of the three infant representatives, and Figures 1 and 2 show the actual infant representatives.

2.2 Subjects

Nine adult volunteers (two females and seven males, ranging in age from 20 to 77 years) subjected both the CRABI-12 and the NCSBS doll to aggressive shaking. Six of the

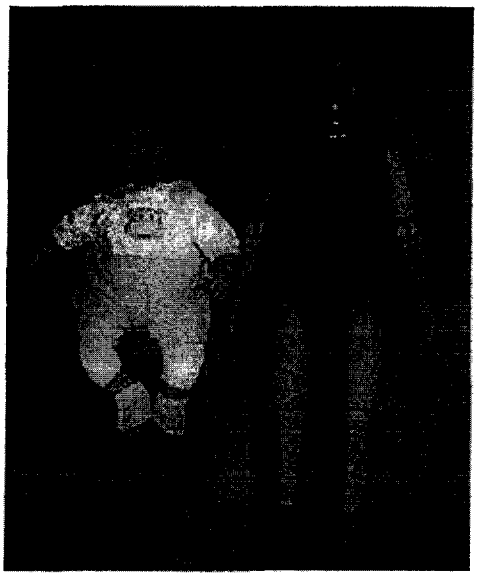


Figure 1: NCSBS demonstration doll (left) and CRABI-12 biofidelic mannequin (right).

Figure 2: CRABI-12 biofidelic mannequin (left) and 7-month-old infant, KL (right).

volunteers also handled the CRABI-12 in various ways to replicate activities of daily living (ADLs) for an infant, such as being burped or rocked.

The actual infant, KL, provided data during the normal course of his day, while playing in his Jumparoo, which is an infant's play toy manufactured by Fisher Price. At the time of the study, he met the specifications in the product's instruction manual, which cautions that it is used "only for a child who is able to hold head up unassisted and who is not able to crawl out."

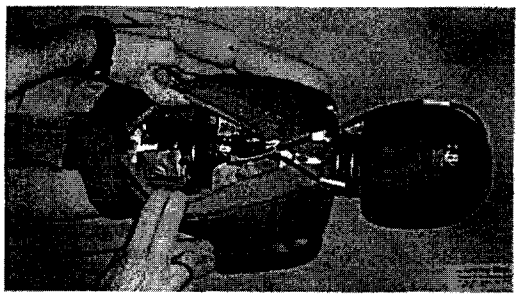


Figure 3: InterSense sensors on head and torso of CRABI-12 mannequin.

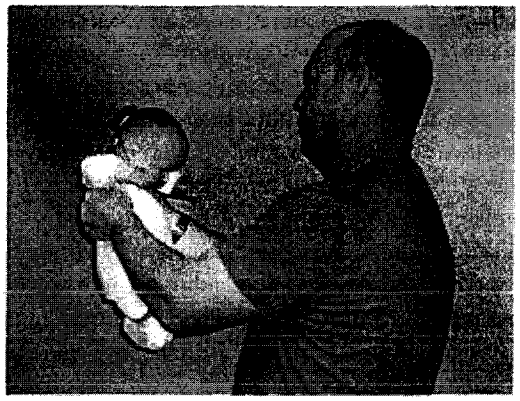


Figure 4: Male subject demonstrating aggressive shaking with NCSBS doll.

2.3 Test protocol

InterSense sensors (InterSense, Inc., Billerica, MA, USA) were secured to the head and torso of the child surrogate, as illustrated in Figure 3 for the CRABI-12 mannequin.

During the trials, the sensors transmitted raw data at 179 Hz per channel—including orientation (yaw, pitch, and roll), quaternion, angular velocity, and linear acceleration—wirelessly to a nearby computer. The sampling rate far exceeds the Niquist frequency for the shaking and pediatric ADL activities investigated.

Data was collected in three sets:

(i) from the NCSBS doll and the CRABI-12 mannequin while being shaken by an adult;

(ii) from the CRABI-12 mannequin during activities of daily living (ADLs), listed later in this section;

(iii) from the human infant at play, treated in the data analysis as an ADL.

2.3.1 Shaking

Nine adult volunteers grasped each of the two infant substitutes, the NCSBS doll and the CRABI-12 mannequin, as illustrated in Figure 4.

Figure 5: Female subject bouncing CRABI mannequin on her knee.

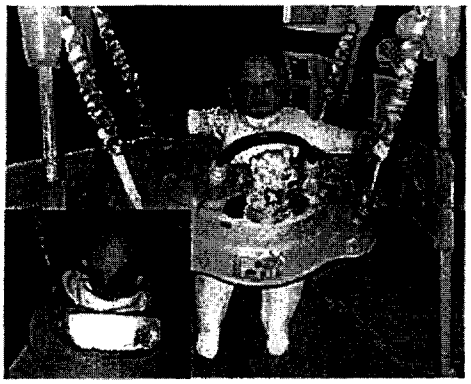


Figure 6: 7-month-old infant, KL, at play in his Fisher Price Jumparoo. Inset: InterSense sensor on back of subject KL's head.

While the sensors transmitted data, the volunteers shook the infant surrogates using three different techniques:

(i) mild shaking, to simulate resuscitative efforts;
(ii) gravity-assisted shaking, where the doll or mannequin was swung forcefully towards the ground, but without impact;
(iii) aggressive, repetitive shaking in the horizontal plane.

Each volunteer shook the infant representatives as hard as he or she could for as long as possible. Most subjects accomplished 10–20 seconds of shaking at 3–5 Hz. Each volunteer repeated the shaking twice, for a total of three trials for each ATD, per subject.

2.3.2 Pediatric activities of daily living

A subset of six adult volunteers, including two females and four males, manipulated the CRABI-12 mannequin during various pediatric ADLs, whilst head motion data was acquired using the InterSense sensors, as previously described. The investigated ADLs included:

(i) pushing the mannequin in a stroller over a smooth surface;
(ii) pushing the mannequin in a stroller over an uneven surface;
(iii) rocking the mannequin in a powered cradle;
(iv) walking on a treadmill at 2.5 mph while holding the mannequin in a baby carrier;
(v) running on a treadmill at 6.5 mph while holding the mannequin in a baby carrier;
(vi) throwing the mannequin into the air and catching it;
(vii) burping the mannequin with a back slap;
(viii) burping the mannequin with an up-and-down shake;
(ix) consoling the mannequin;
(x) bouncing the mannequin on a knee, as illustrated in Figure 5;
(xi) hitching the mannequin up onto the hip;
(xii) swinging the mannequin back and forth.

As in the shaking trials, each volunteer performed each activity three times. Only the CRABI-12 mannequin was used for these trials.

2.3.3 Infant playing

InterSense sensors were attached to the posterior aspect of the head of the 7-month-old male infant, KL, before he began one of his favorite activities, jumping in a commercially available jumping toy. Researchers collected data from KL's bouncing in 37 separate trials on two non-consecutive days, one week apart. The average minimum duration across trials was about 30 seconds.

Figure 6 illustrates the subject ready to jump.

2.4 Analysis

Using MatLab (The MathWorks, Natick, MA, USA), the investigators performed Fast Fourier Transform (FFT) analysis to isolate environmental noise data, which was removed using a phaseless 4th-order Butterworth low-pass filter, with a cut-off frequency of 50 Hz. Angular accelerations were derived, root-mean-square (RMS) values were calculated, and Head Injury Criterion (HIC-15) was computed according to (1), where HIC-15 is calculated with a period of less than 15 ms. A HIC-15 value of 390 is estimated to represent a risk of a severe head injury based on studies with the CRABI 6-month-old biofidelic mannequin [39]:

$$\mathrm{HIC} = \left[\frac{1}{t_2 - t_1}\int_{t_1}^{t_2} a\,dt\right]^{2.5}(t_2 - t_1), \tag{1}$$

where a is a resultant head acceleration, $t_2 - t_1 < 15$ ms and t_2, t_1 were selected so as to maximize HIC.

	CRABI Resuscitative	CRABI Gravity assist	CRABI Aggressive shaking	NCSBS Resuscitative	NCSBS Gravity assist	NCSBS Aggressive shaking
AngDisp (deg)	50.8 (1.4)	121.9 (2.4)	120.8 (2.7)	71.6 (5.9)	128.6 (10.2)	167.4 (4.4)
AngVel RMS (rads-1)	12.5 (0.4)	24.3 (1.5)	25.5 (0.6)	12.5 (1.2)	35.7 (2.4)	34.6 (0.7)
AngAccel RMS (rads-2)	364.6 (20.8)	581.5 (57.2)	1068.3 (38.9)	502.9 (68.3)	995.4 (219.7)	1587.0 (79.0)
LinAccel RMS (g)	3.2 (0.1)	7.2 (0.3)	7.6 (0.2)	3.6 (0.4)	9.8 (0.1)	9.9 (0.2)
HIC-15	0.3 (0.03)	2.5 (0.2)	2.6 (0.1)	0.6 (0.2)	5.0 (0.1)	4.9 (0.3)

Table 2: Peak magnitudes, averaged across trials, recorded during different shaking techniques using the two infant surrogates (standard error of the mean in parentheses).

	Stroller (uneven)	Running (6.5 mph)	Throw in air & catch	Burping (back slap)	Bounce on knee	KL Jumparoo
AngDisp (deg)	14.2 (0.6)	59.3 (2.4)	58.6 (1.5)	12.0 (0.7)	44.2 (4.6)	77.8 (2.2)
AngVel RMS (rads-1)	2.9 (0.1)	8.3 (0.4)	7.8 (0.4)	1.3 (0.05)	6.5 (0.3)	15.6 (0.7)
AngAccel RMS (rads-2)	175.1 (0.8)	241.7 (8.8)	258.8 (19.5)	101.1 (6.0)	169.3 (7.5)	954.4 (35.0)
LinAccel RMS (g)	3.1 (0.05)	4.3 (0.2)	3.7 (0.2)	1.0 (0.1)	2.7 (0.2)	3.4 (0.1)
HIC-15	0.2 (0.02)	0.7 (0.05)	0.5 (0.05)	< 0.1 (0.02)	0.2 (0.03)	0.4 (0.02)

Table 3: Peak magnitudes, averaged across trials, during a selection ADLs using the CRABI biofidelic mannequin (standard error of the mean in parentheses).

3 Results

Table 2 reports the peak values, averaged across multiple trials and subjects, for each of the three shaking techniques performed with each of the two infant surrogates, the CRABI-12 mannequin and the NCSBS doll.

Table 3 reports the peak values, averaged across multiple trials and subjects, for a selection of pediatric ADLs, including the infant KL playing in his Jumparoo.

Figure 7 graphically illustrates the head kinematics during both infant shaking and pediatric activities of daily living. Values represent peak angular acceleration of the head, averaged across subjects and trials.

An analysis of results was conducted using SAS statistical analysis software (SAS Institute Inc., Cary, NC, USA). Findings denote that peak angular head accelerations recorded from the infant during bouncing in the Jumparoo (954.4 rad/s^2) are statistically indistinguishable at $P \leq .05$ from those recorded during aggressive shaking of the CRABI biofidelic mannequin (1068.3 rad/s^2).

Investigators also noted that values recorded during shaking of the NCSBS demonstration doll are approximately 50% higher than those recorded during shaking of the CRABI mannequin.

Most notably, even the results for aggressive shaking of the NCSBS doll are 84% below the scientifically accepted threshold for brain injury from angular acceleration.

4 Limitations of the present study

Any research using ATDs is limited by the biofidelity of the models employed.

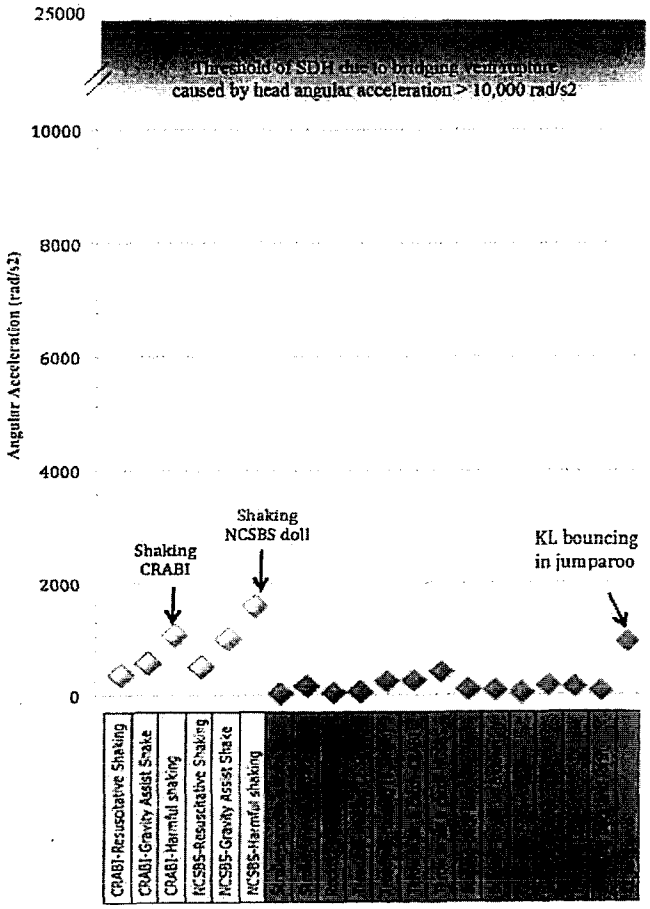


Figure 7: Head angular acceleration, in radians per second squared, during shaking (yellow) versus pediatric ADLs (blue).

Prange et al., 2003, reported that their ATD neck was specifically modified for the "worst-case scenario of no resistance provided by the neck, so that [they] could ascertain the greatest possible velocities and accelerations that can be generated by these mechanisms" [36]. The neck in their model was a single, linear metal hinge that connected the head to the torso, allowing free motion in one orientation only, neck flexion and extension in the sagittal plane. The highest-magnitude angular accelerations—2600 rad/s^2—were recorded when the model's head hit its own chest and back. Without trying to replicate the infant neck precisely, the Prange team concluded that "there are no data demonstrating that maximal angular velocities and maximal angular accelerations experienced during shaking and inflicted impact against foam cause SDH or TAI [traumatic axonal injury]".

Also in 2003, Cory and Jones reported that shaking their preliminary model produced a series of chin-to-chest and occiput-to-back impacts. Their discussion leaves open the question of how well this model reflects the neck of a human infant [11].

As illustrated in Figure 8, which was extracted from a high-speed digital video, angular displacement of the CRABI-12 neck in the sagittal plane shows a possible endpoint impact during shaking.

As in previous studies, angular accelerations of the ATD head during shaking reached maximum values at the end points of angular displacement.

When Wolfson et al. conducted calculated shaking simulations with ATDs of varying neck stiffnesses, only the models with "end-stop-type neck stiffness characteristics" produced values above predicted injury levels. More hinges added to the neck model to improve biofidelity produced lower head accelerations. The authors discounted the likelihood of impact with the back as the source of SBS symptoms with the observation "if violent impact of the head against the torso were the mechanism of intracranial injury in SBS, it is likely that findings such as bruising of the chin, chest, back and occiput would be reported" [40].

The infant KL was not photographed using a high-speed camera. The videos show no apparent contact between the child's head and body. More research is needed to investigate tissue properties and safe range of motion of the infant neck.

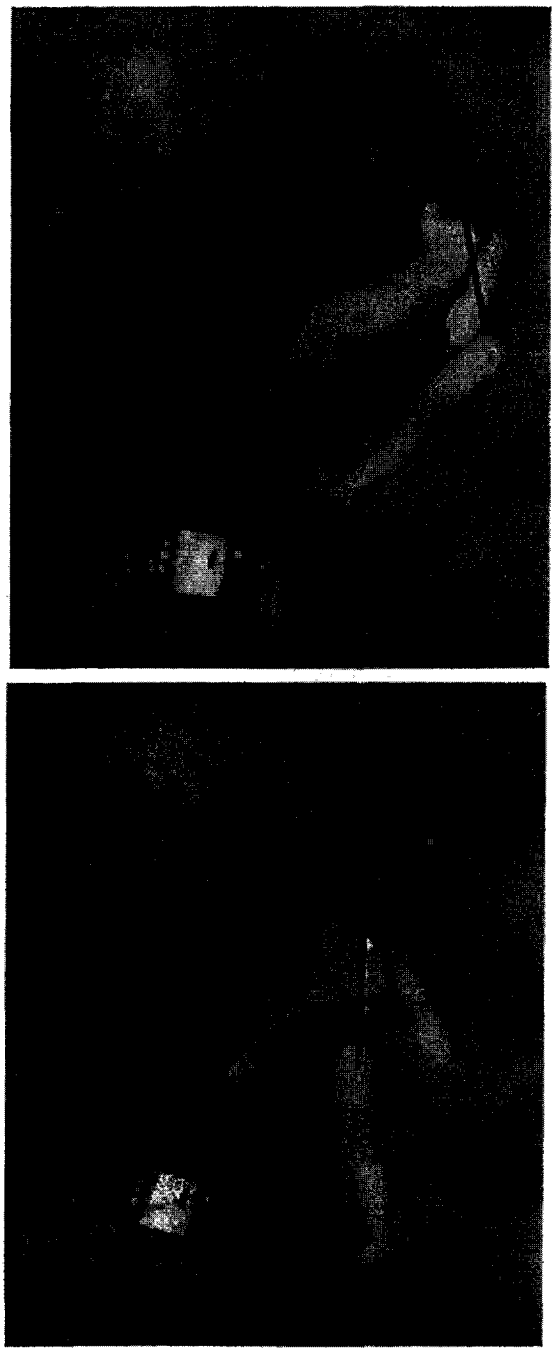


Figure 8: CRABI neck in the sagittal plane shows possible endpoint impact during shaking.

5 Discussion

It has been assumed for decades that aggressive manual shaking, with or without impact, produces the characteristic "triad" of SBS symptoms (subdural hemorrhages, cerebral edema/brain swelling and retinal hemorrhages) in an infant. This model is based on the hypothesis that uncontrolled motion of the infant head during shaking causes the damage directly, by tearing bridging veins to produce subdural hematoma, stretching neurons to produce diffuse axonal injury, and causing vitreous traction on the retina to produce retinal hemorrhages, schisis and folds.

Our study, however, like others before it, demonstrates that an adult's shaking of an infant surrogate does not even approach the angular accelerations generally accepted as a minimum threshold for infant SDH and DAI. These repeated experimental results undermine the fundamental thinking behind the abusive shaking hypothesis.

Type	Variations
Trauma	Accidental, falls and otherwise
	Inflicted, with impact or otherwise
Prenatal, perinatal, and pregnancy-related conditions causing intra-cranial hemorrhage	Intrauterine trauma, including abruptio placenta
	Thrombocytopenia
	Eclampsia and preeclampsia
	Chorioamnionitis
	Multiple pregnancies
	Hemolytic disease of newborn
	Prematurity
	Germinal matrix hemorrhage
Trauma at delivery	Abnormal presentation and uterine abnormalities
	Prolonged labor
	Forceps delivery
	Vacuum extraction
	Manual manipulation of the fetus
	Chemically assisted labor (pitocin drips, misoprostol (Cytotec))
Congenital malformations	Chiari malformations
	Arteriovenous malformations
	Aneurysm
	Osler-Weber syndrome
	Arachnoidal cyst
	Hydrocephalus, including extra-axial fluid collections
	Meningocele
	Syringomyelia
Venous and sinus thrombosis	Blood coagulation defects
	Leukemia
	Nephrotic syndrome
	Local infection
	Dehydration
	Hypernatremia
	Trauma induced central venous thrombosis
Genetic and metabolic conditions	Hemoglobinopathies, sickle cell disease
	Osteogenesis imperfecta
	Ehlers-Danlos Syndrome
	Von Recklinghausen's disease
	Tuberous sclerosis
	Marfan syndrome
	Menkes disease
	Polycystic kidney disease
	Glutaric aciduria
	Galactosemia
	Homocystinuria
	Alpha 1-antitrypsin deficiency
	Hemophagocytic lymphohistiocytosis, primary or secondary
	Vitamin D deficiency during pregnancy
	Others
Bleeding and/or coagulation disorders	Vitamin K deficiency
	Vitamin C deficiency
	Hemophilia A
	Hemophilia B
	Factor V deficiency
	Factor XII deficiency
	Factor XIII deficiency
	Protein S deficiency
	Protein C deficiency
	Von Willebrand disease
	Dysfibrinogenemia or hypofibrinogenemia
	Thrombocytopenic purpura
	Disseminated intravascular coagulation especially with infection or neoplasm
	Cirrhosis
	Inhibitors to clotting factors, including the following:
	– lupus erythematosus,
	– antiphospholipid antibody syndrome,
	– others
Infection	Meningitidis associated with numerous bacterial pathogens, including
	Streptococcus pneumoniae,
	Haemophilus influenzae,
	and Neisseria meningitidis
	Herpes encephalitis
	Cytomegalovirus encephalitis
	Infections in sinuses and/or ears
	Tonsillitis
	Toxoplasmosis
	Undetermined
Ischemic-hypoxic encephalopathy	
Vascular abnormalities	Moyamoya disease
	Kawasaki disease
	Dissecting vasculopathy
	Others
Neoplasms	Medulloblastoma and primitive neuroectodermal tumor
	Neuroblastoma
	Wilms tumor
	Leukemia
	Lymphoma
	Choroid plexus papilloma
	Xanthogranuloma
	Others
Medical interventions	Anticoagulation
	Craniotomy
	Spinal tap
	Spinal anesthesia
	Epidural anesthesia
	Subdural taps
	Intrathecal injection
	Shunts for hydrocephalus
	Placement of monitors
	Intravenous lines
	Antineoplastic therapy
	Anti-cold medications
Non-pharmaceutical toxins	Cocaine
	Lead
	Other

Table 4: Differential diagnosis for intracranial bleeding and cerebral edema.

The triad, meanwhile, is often found in children who present with seizures, which can interrupt breathing. Decreased oxygen supply can itself trigger cerebral edema. Tissue studies have concluded that brain damage in inflicted head injury results more from hypoxic-ischemic injury than from DAI [19,22,29].

Bridging vein rupture, meanwhile, is unlikely to be the source of low-volume intracranial hemorrhages in infants. Returning blood from the superior portions of the cerebral hemispheres flows through 5–8 pairs of bridging veins into the superior sagittal sinus. Given a blood flow of 50 mL per minute for every 100 grams of brain, each of these bridging veins would be expected to carry at least 5–10 mL of blood per minute. If a vein were to rupture, large volumes of subdural bleeding would be expected. However, autopsy findings in children report collections ranging from 1 to 80 mL, with 75% less than 25 mL and 50% less than 10 mL [33]. These small, thin films of subdural blood

seem clinically insignificant and inconsistent with bridging vein rupture. Researchers have suggested that in pediatric cases with minor hemorrhagic collections the blood may emanate from intra-dural vessels, rather than bridging vein rupture [32,38]. If the source of this blood is the dura, then biomechanical studies of bridging vein tolerances may not apply, yet these minor hemorrhagic collections frequently play a large role in legal proceedings.

Physicians have long recognized that the same clinical presentation can have more than one possible cause. Diagnosticians are therefore trained to apply adductive reasoning, ruling in and ruling out causes systematically, in what is known as differential diagnosis. The doctor hypothesizes the most likely cause: if a patient does not respond to treatment, or subsequent findings fail to confirm or even contradict the working diagnosis, other potential causes must be considered.

A diagnosis of SBS, unfortunately, can prematurely terminate the search for other possible causes of an infant's symptoms. A fundamental tenet of the classic SBS hypothesis is that abusive head trauma can occur in the absence of any other signs of abuse: no abrasions, no bruises, no neck or spinal cord damage, only the pattern of intracranial bleeding and swelling. This criterion—no signs of trauma—also applies to a host of other causes of intracranial bleeding and cerebral edema. Table 4, a list of current known causes, is adapted from a chapter in a reference text [37] and a medical journal article [12].

Shaken baby syndrome prosecutions, perhaps uniquely, rest primarily if not entirely on medical opinion. The same papers that established professional guidelines for identifying SBS also specified that, in cases of serious injury, the symptoms of an aggressive shaking would become apparent immediately after the assault [2,7]. With this nuance in place, the testimony of doctors is used to establish (1) that a crime was committed, (2) what actions constituted the crime and (3) when the crime occurred. Police officers who receive this information from a doctor see their jobs as to establish who was with the baby when the symptoms emerged, and then build a case against that person.

To make a diagnosis this powerful, a physician must rely on only the most solid evidence. Although the original SBS hypothesis has enjoyed decades of general acceptance, results from repeated biomechanical studies continue to undermine the reliability of the basic model, while timing of the symptoms also remains controversial [23,26] and researchers in other specialties continue to raise questions about various aspects of the classic model [4,20,21,30].

6 Conclusions

This study demonstrates that angular acceleration of the head during aggressive shaking of the CRABI biofidelic mannequin (1068.3 rad/s^2) is statistically indistinguishable ($P \leq .05$) from angular head kinematics experienced by a 7-month-old infant fervently playing in his Jumparoo (954.4 rad/s^2). Other pediatric ADLs, such as being burped or bounced on a knee, are clearly negligible. Furthermore, measured angular accelerations fall 84% below the scientifically accepted biomechanical threshold for bridging-vein rupture of 10,000 rad/s^2.

Although shaking an infant or toddler in anger is clearly ill advised and potentially unsafe, our data indicate that neither aggressive nor resuscitative shaking is likely to be a primary cause of diffuse axonal injury, primary retinal hemorrhage, schisis or folds, or subdural hematoma in a previously healthy infant.

Future research will investigate a systematic protocol for evaluating biomechanical indices associated with falls from different heights and orientations onto various surfaces.

References

[1] M. Allen, I. Weir-Jones, D. Motiuk, K. Flewin, R. Goring, R. Kobetitch, et al., *Acceleration perturbations of daily living. A comparison to 'whiplash'*, Spine, 19 (1994), 1285–1290.

[2] American Academy of Pediatrics: Committee on Child Abuse and Neglect, *Shaken baby syndrome: rotational cranial injuries-technical report*, Pediatrics, 108 (2001), 206–210.

[3] F. A. Bandak, *Shaken baby syndrome: A biomechanics analysis of injury mechanisms*, Forensic Sci Int, 151 (2005), 71–79.

[4] P. D. Barnes, *Imaging of nonaccidental injury and the mimics: issues and controversies in the era of evidence-based medicine*, Radiol Clin North Am, 49 (2011), 205–229.

[5] R. W. Block, *SBS/AHT 2010: What we know, what we must learn, what we must do to move forward*, in Eleventh International Conference on Shaken Baby Syndrome/Abusive Head Trauma, National Center On Shaken Baby Syndrome, Atlanta, GA, 2010.

[6] J. Caffey, *On the theory and practice of shaking infants: Its potential residual effects of permanent brain damage and mental retardation*, Am J Dis Child, 124 (1972), 161–169.

[7] M. E. Case, M. A. Graham, T. C. Handy, J. M. Jentzen, and J. A. Monteleone, *Position paper on fatal abusive head injuries in infants and young children*, Am J Forensic Med Pathol, 22 (2001), 112–122.

[8] D. L. Chadwick, R. H. Kirschner, R. M. Reece, L. R. Ricci, R. Alexander, M. Amaya, et al., *Shaken baby syndrome—a forensic pediatric response*, Pediatrics, 101 (1998), 321–323.

[9] C. W. Christian and R. Block, *Abusive head trauma in infants and children*, Pediatrics, 123 (2009), 1409–1411.

[10] Commonwealth v Ann Power, 2005, *Report to the Middlesex County District Attorney Office Cambridge Massachusetts by Carole Jenny*. December 29, 2005.

[11] C. Z. Cory and B. M. Jones, *Can shaking alone cause fatal brain injury? A biomechanical assessment of the Duhaime shaken baby syndrome model*, Med Sci Law, 43 (2003), 317–333.

[12] T. J. David, *Non-accidental head injury—the evidence*, Pediatr Radiol, 38 (2008), S370–S377.

[13] B. Depreitere, C. Van Lierde, J. V. Sloten, R. Van Audekercke, G. Van der Perre, C. Plets, et al., *Mechanics of acute subdural hematomas resulting from bridging vein rupture*, J Neurosurg, 104 (2006), 950–956.

[14] K. Desantis Klinich, G. M. Hulbert, and L. W. Schneider, *Estimating infant head injury criteria and impact response using crash reconstruction and finite element modeling*, Stapp Car Crash J, 46 (2002), 165–194.

[15] A. C. Duhaime, C. W. Christian, L. B. Rorke, and R. A. Zimmerman, *Nonaccidental head injury in infants—the "shaken-baby syndrome"*, N Engl J Med, 338 (1998), 1822–1829.

[16] A. C. Duhaime, T. A. Gennarelli, L. E. Thibault, D. A. Bruce, S. S. Margulies, and R. Wiser, *The shaken baby syndrome. A clinical, pathological, and biomechanical study*, J Neurosurg, 66 (1987), 409–415.

[17] J. M. Duncan, *Laboratory note: On the tensile strength of the fresh adult foetus*, Br Med J, 2 (1874), 763–764.

[18] J. R. Funk, S. M. Duma, S. J. Manoogian, and S. Rowson, *Biomechanical risk estimates for mild traumatic brain injury*, Annu Proc Assoc Adv Automot Med, 51 (2007), 343–361.

[19] J. F. Geddes, A. K. Hackshaw, G. H. Vowles, C. D. Nickols, and H. Whitwell, *Neuropathology of inflicted head injury in children. I. Patterns of brain damage*, Brain, 124 (2001), 1290–1298.

[20] J. F. Geddes and J. Plunkett, *The evidence base for shaken baby syndrome*, Br Med J, 328 (2004), 719–720.

[21] J. F. Geddes, R. C. Tasker, A. K. Hackshaw, C. D. Nickols, G. G. Adams, H. Whitwell, et al., *Dural haemorrhage in non-traumatic infant deaths: does it explain the bleeding in 'shaken baby syndrome'?*, Neuropathol Appl Neurobiol, 29 (2003), 14–22.

[22] J. F. Geddes, G. H. Vowles, A. K. Hackshaw, C. D. Nickols, I. S. Scott, and H. Whitwell, *Neuropathology of inflicted head injury in children. II. Microscopic brain injury in infants*, Brain, 124 (2001), 1299–1306.

[23] M. G. Gilliland, *Interval duration between injury and severe symptoms in nonaccidental head trauma in infants and young children*, J Forensic Sci, 43 (1998), 723–725.

[24] A. N. Guthkelch, *Infantile subdural haematoma and its relationship to whiplash injuries*, Br Med J, 2 (1971), 430–431.

[25] B. Harding, R. A. Risdon, and H. F. Krous, *Shaken baby syndrome*, Br Med J, 328 (2004), 720–721.

[26] R. W. Huntington III, *Symptoms following head injury*, Am J Forensic Med Pathol, 23 (2002), 105–106.

[27] N. G. Ibrahim and S. S. Margulies, *Biomechanics of the toddler head during low-height falls: an anthropomorphic dummy analysis*, J Neurosurg Pediatr, 6 (2010), 57–68.

[28] C. Jenny, *Junk Medical Science in the Courtroom*. Rhode Island Hospital Pediatric Grand Rounds. Providence, RI (July 23, 2010), http://lifespan.mediasite.com/mediasite/Viewer/?peid=d237bed531df42e49223ccdb685c48741d.

[29] A. M. Kemp, N. Stoodley, C. Cobley, L. Coles, and K. W. Kemp, *Apnoea and brain swelling in non-accidental head injury*, Arch Dis Child, 88 (2003), 472–476.

[30] J. E. Leestma, *Case analysis of brain-injured admittedly shaken infants: 54 cases, 1969–2001*, Am J Forensic Med Pathol, 26 (2005), 199–212.

[31] J. F. Luck, R. W. Nightingale, A. M. Loyd, M. T. Prange, A. T. Dibb, Y. Song, et al., *Tensile mechanical properties of the perinatal and pediatric PMHS osteoligamentous cervical spine*, Stapp Car Crash J, 52 (2008), 107–134.

[32] J. Mack, W. Squier, and J. T. Eastman, *Anatomy and development of the meninges: implications for subdural collections and CSF circulation*, Pediatr Radiol, 39 (2009), 200–210.

[33] D. K. Molina, A. Clarkson, K. L. Farley, and N. J. Farley, *A review of blunt force injury homicides of children aged 0 to 5 years in Bexar County, Texas, from 1988 to 2009*, Am J Forensic Med Pathol, (2011).

[34] J. Ouyang, Q. Zhu, W. Zhao, Y. Xu, W. Chen, and S. Zhong, *Biomechanical assessment of the pediatric cervical spine under bending and tensile loading*, Spine, 30 (2005), E716–E723.

[35] M. Prange, W. Newberry, T. Moore, D. Peterson, B. Smyth, and C. Corrigan, *Inertial neck injuries in children involved in frontal collisions*. Society of Automotive Engineers, SAE 2007-01-1170 (presented at the 2007 SAE World Congress, Detroit, MI), 2007.

[36] M. T. Prange, B. Coats, A. C. Duhaime, and S. S. Margulies, *Anthropomorphic simulations of falls, shakes, and inflicted impacts in infants*, J Neurosurg, 99 (2003), 143–150.

[37] A. Sirotnak, *Medical disorders that mimic abusive head trauma*, in Trauma in Infants and Children, L. S. Frasier, R. Alexander, K. Rauth-Farley, and R. N. Parrish, eds., GW Medical Publishing, St. Louis, 2006, 191–226.

[38] W. Squier, E. Lindberg, J. Mack, and S. Darby, *Demonstration of fluid channels in human dura and their relationship to age and intradural bleeding*, Childs Nerv Syst, 25 (2009), 925–931.

[39] C. Van Ee, B. Moroski-Browne, D. Raymond, K. Thibault, W. Hardy, and J. Plunkett, *Evaluation and refinement of the CRABI-6 anthropomorphic test device injury criteria for skull fracture*, in Proceedings of the ASME 2009 International Mechanical Engineering Congress & Exposition, Lake Buena Vista, FL, 2009.

[40] D. R. Wolfson, D. S. McNally, M. J. Clifford, and M. Vloeberghs, *Rigid-body modelling of shaken baby syndrome*, Proc Inst Mech Eng H, 219 (2005), 63–70.

[41] L. Zhang, K. H. Yang, and A. I. King, *A proposed injury threshold for mild traumatic brain injury*, J Biomech Eng, 126 (2004), 226–236.

EXHIBIT O

Neurol Med Chir (Tokyo) 46, 57～61, 2006

Shaken Baby Syndrome: An Odyssey

Ronald H. USCINSKI

Department of Neurosurgery, George Washington University Medical Center, Washington, D.C., U.S.A.; Department of Neurosurgery, Georgetown University Medical Center, Washington, D.C., U.S.A.; Potomac Institute for Policy Studies, Arlington, Virginia, U.S.A.

Abstract

Shaken baby syndrome is evaluated in the context of its historical evolution and its veracity in referring to causal injury mechanisms. A rational assessment of the injury causation and consequent pathological states associated with the syndrome is presented. It is now evident that shaken baby syndrome evolved as a result of a faulty application of scientific reasoning and a lack of appreciation of mechanisms of injury. A brief explanation of the commonly understood usage and interface of scientific methodology and reasoning as applied to clinical medicine is given.

Key words: shaken baby syndrome, injury biomechanics, subdural hematoma

Introduction

Shaken baby syndrome is characterized as a constellation of clinical findings including subdural bleeding, retinal hemorrhages, and associated fractures of the extremities or ribs, with no external evidence of cranial trauma. This widely proclaimed yet still hypothetical supposition has become a virtually unquestioned assumption nowadays as a modality for causing inflicted intracranial injury in infants. In 1997 the author was asked to review the case of a child fatally injured supposedly by shaking, and in doing so researched the entire body of literature referencing the so-called "shaken baby syndrome." This article is a product of that effort, and in a sense represents an intellectual "odyssey."[15] The paper is divided into three parts. The first places the syndrome in the historical perspective of the original papers providing the initial description of shaken baby syndrome. The second part gives a brief discussion of the physical laws of motion governing injury to relevant body structures, and encompasses a brief overview of the known pathophysiology of certain relevant forms of intracranial injury. The final section discusses the methodology of scientific reasoning and experimentation and its significance in the context of the immediate subject of the so-called inflicted shaking injury and the broader context of observing and understanding phenomena in our physical world.

Historical Perspective

The quantity of articles dealing with shaking as a putative mechanism for inflicting intracranial injury in infants has increased significantly since the 1970s when the original description of shaking first appeared in a paper published in The British Medical Journal in 1971 by Guthkelch.[8] Caffey,[2,3] who is generally credited with identifying and characterizing injuries to infants by shaking, published extensively on the subject thereafter. However, it is in the

---

This excellent paper was presented on May 25, 2005 at the 33rd Annual Meeting of The Japanese Society for Pediatric Neurosurgery in Nara, chaired by Professor Toshisuke Sakaki. All of the audience was greatly impressed by this new and unique concept of the so-called Shaken Baby Syndrome.

Shaken Baby Syndrome has now become a social issue in Japan and neurosurgeons are very much involved in its management. The topic was quite timely, so we thank Dr. Sakaki for selecting this paper as a special lecture for us.

Dr. Ronald H. Uscinski and I were residents together in neurosurgery at the Georgetown University Hospitals, Washington, D.C. in the early 1970s. I am very proud that such an excellent paper was produced in Washington, D.C. and presented in Nara.

Akira YAMAURA, M.D. (Advisory Board of *Neurologia medico-chirurgica*)
President, Chiba College of Health Sciences, Chiba

Guthkelch article that shaking is invoked specifically as a mechanism for causing intracranial injury in infants with no external evidence of cranial trauma. Guthkelch hypothesized that "It seems clear that the relatively large head and puny neck muscles of the infant must render it particularly vulnerable to whiplash injury in this sort of situation," and moreover that "the rotation-acceleration strains on the brain would tend to occur fairly symmetrically also, in an anteroposterior direction. This may be the same reason why the infantile subdural haematoma is even more often bilateral..."

It is significant that as a mechanical justification for invoking shaking as a mechanism for causing intracranial injury in infants, the original and subsequent authors all reference a single paper by Ommaya et al., published in 1968.[16] In this paper, Ommaya, a neurosurgeon studying head injury and building on earlier work by Holbourn,[9,10] attempted to quantify experimentally the rotational acceleration necessary to cause intracranial injury via a whiplash in rhesus monkeys. The animals in Ommaya's experiment were placed in a contoured fiberglass chair with the head free to rotate; the chair was mounted on wheels and placed on tracks; and the apparatus was systematically impacted with a piston to simulate accelerations analogous to rear-end motor vehicle collisions, with the entire event photographed using a high-speed camera. Ommaya found intracranial injury in 18 of the animals, with concomitant neck injury in 11 of the 18. The purely isolated concept of rotational acceleration of sufficient magnitude to cause intracranial injury without impact and therefore without external evidence of injury was seized upon qualitatively by Guthkelch,[8] Caffey,[2,3] and others as the explanation for hitherto unexplained intracranial injury in infants. This concept was hypothesized and put forth by them as being the result of manual shaking.

Hence, the Ommaya paper emerges as the sole source of experimental data from which the initial hypothetical shaking mechanism was drawn. Significantly, Ommaya was actually attempting to quantify the requisite rotational acceleration necessary to cause head injury via whiplash movement of the head in humans during rear end motor vehicle collisions, with attendant vehicular impact. *He never addressed the question as to whether human beings can shake infants with enough force to produce the acceleration necessary to cause intracranial injury.* It is also significant that neither Guthkelch,[8] Caffey,[2,3] nor any subsequent investigators who have sought to identify and characterize ostensible shaking injuries to the infant head ever asked whether the infant torso and neck anatomy, quite different physiologically from that of the rhesus monkey and of the "non-infant" human, could withstand such shaking. Nonetheless, the mechanism of shaking and the so named syndrome gained immediate acceptance and enormously widespread popularity, with no real investigation or even question as to its scientific validity.

The stage was set; the shaking hypothesis rapidly engendered numerous articles purporting to accept or validate the hypothesis. Ratification within the medical community was based principally on anecdotal reports and case studies. The nearly simultaneous establishment in the United States of the Mandated Reporting Laws[4] plus the emerging litigious atmosphere encompassing clinical medicine in America in effect rendered the medical reporting of all cases of even remotely suspected child abuse absolutely compulsory.

The combination of these factors, plus an unspoken, unproved, but increasingly pervasive assumption that all unexplained injuries in children were to be regarded as inflicted injuries, provided a new paradigm for a self-fulfilling prophecy. The hypothesis rapidly metamorphosed to a syndrome; its acceptance expanded exponentially, and "shaken baby" became a term synonymous and ultimately identical with intentional child abuse.

Injury Biomechanics

The causes of trauma obey the laws of injury biomechanics. These laws come from the generalized laws that govern motion, deformation, and forces existing in our universe. An example of one of these universal laws is Newton's second law of motion. Newton's second law governs the relationship between mass, acceleration, and force. In other words, given a mass such as a head, the acceleration of such a mass is governed by Newton's second law when there is a change in velocity divided by a change in time.

In 1943 the physicist Holbourn published a laboratory investigation of traumatic brain injury.[9] Holbourn understood that the deformable brain was incompressible, hypothesized a rotational acceleration level beyond which injury would occur, and that a smaller mass of brain would require larger rotational acceleration. Ommaya himself alluded to this point in his paper,[16] although this seems not to have been recognized by Guthkelch,[8] Caffey,[2,3] and others. In 1987 Duhaime et al.,[7] using available data on scaled injury thresholds, demonstrated that shaking a mechanical model to cause intracranial injury in the form of concussion, subdural hematoma, and diffuse axonal injury, failed to reach such

thresholds. The model used three different examples of the infant neck in order to reproduce mobility. None of these examples addressed the potential for structural failure of the neck. Following the same line of thought from 1987, two of the same authors repeated the experiment,[17] again using a model not addressing neck injury mechanisms, again focusing on rotational accelerations as the mechanism for causing intracranial injury as transmitted through the infant neck, and again demonstrating requisite accelerations to be not achievable by manual shaking.

While the above articles addressed experimentally the impossibility of causing intracranial injury in infants by manual shaking, no work addressed the potential consequence of such shaking activity on the infant neck, the critical link between the torso where the physical act of shaking is initiated, and the infant head where the injury is hypothesized to occur. Bandak precisely addressed this question in a quantitative manner in 2005.[1] In focusing attention on the infant neck, and demonstrating thereby that any transmission of forces generated by shaking the infant torso must necessarily be transmitted through the underdeveloped infant neck to the disproportionately large head, Bandak showed clearly that cervical spinal cord or brainstem injury in the infant would occur at significantly lower levels of shaking accelerations than those purported in the shaken baby syndrome literature as a cause of subdural hematomas. It is now clear that if an infant is subjected to shaken baby syndrome accelerations one should expect to see injury in the infant neck before it is seen in the head. Moreover, such injury should include injury to the cervical spinal cord and brainstem, obviously with the expected accompanying clinical picture.

Based on the above cited material, it is clear that the hypothetical mechanism of manually shaking infants in such a way as to cause intracranial injury is based on a misinterpretation of an experiment done for a different purpose, and contrary to the laws of injury biomechanics as they apply specifically to the infant anatomy. Finally, manual shaking of an infant, if injurious, should result in an entirely different injury biomechanically, physiologically, and clinically, than hypothesized in 1971.

The "Unexplained Head Injury"

With regard to the cardinal aspect of inflicted injury by the hypothetical shaking mechanism, the "unexplained head injury," the following salient points are worthy of consideration. First, the so-called trivial head impact occurring after a fall of an apparently short distance is believed in most instances to be an innocuous event. In fact this is not so. The free fall velocity from as little as 3 feet results in the equivalent of greater than 9 miles per hour against a hard surface, or more than twice the skull fracture energy for an infant, again as demonstrated by Bandak.[1] Cadaver testing demonstrated skull fractures in infant specimens in every case when dropped from a height of 3 feet (84 cm), or the height of a changing table, onto a firm or hard surface. Fractures in the thin parietal bone were reliably produced when specimens were dropped even onto a softly cushioned surface.[18-20]

Therefore, while the majority of such falls may be seen superficially as innocuous, there exists demonstrably proven potential for serious injury. This is compounded given the potential physiologic response to such injury including vomiting, aspiration, seizing, or other airway compromise with attendant potential for hypoxia, all further complicating the clinical picture. One concludes that rather than resorting to simple generalization, each case warrants careful and individual evaluation.

The second point is that subdural hematoma has hitherto been regarded as a cardinal sign in infants of inflicted injury. While acute subdural hematoma is typically seen after an obvious fall with impact, it must be differentiated from chronic subdural hematoma. Most neurosurgeons treating adult or pediatric patients are aware that chronic subdural hematoma certainly started acutely, but by definition its presence was either not recognized or its significance was not appreciated at the time of injury. This need not imply an intentional injury, and it is a matter worthy of some reflection that intentional injury is rarely diagnosed or even considered in an adult presenting with a chronic subdural hematoma. Yet the same injury with the same pathophysiology and the same pathologic anatomy is nowadays presumed to be intentional in the infant. The scientific grounding for this presumption remains unclear.

Some additional observations are noteworthy here. First, it has long been known among clinical neurosurgeons operating on patients with chronic subdural hematomas that at surgery fresh blood may be found in addition to the older blood comprising the hematoma. Second, it has also been demonstrated experimentally that chronic subdural hematomas enlarge by rebleeding from the neovascular membrane[11-13,21] and that this bleeding has been shown to occur without accompanying trauma. Therefore, at clinical presentation a chronic subdural hematoma may exhibit fresh blood and this may be

mistakenly diagnosed as evidence of recent injury. Lastly, it is known that intracranial hemorrhage may occur even after an apparently uneventful vaginal birth,[5] and it is also well known that a chronic subdural hematoma with well-developed outer and inner membranes is at least several weeks, or even months, old.

The above observations lead one to conclude that for an infant presenting with ostensibly unexplained intracranial bleeding with or without external evidence of injury under given circumstances, accidental injury from a seemingly innocuous fall, perhaps even a remote one, or even an occult birth injury, must be considered before assuming intentional injury.

A recent paper by Donohoe[6] attempted to evaluate the available medical scientific evidence published from 1966 to 1998 wherein internationally accepted methods were used to determine the degree of confidence that accrues to claims regarding the condition termed "shaken baby syndrome." He concluded that some 32 years of cumulative material yielded inadequate scientific evidence to establish a firm conclusion on most aspects of causation, diagnosis, treatment, or any other matters pertaining to shaken baby syndrome. Donohoe's assessment focused on the methods and quality of the actual research. The scientific status of the syndrome itself was not addressed; rather it was the methodology supporting its validity that was found to be insufficient. Another paper by Leestma[14] searched all of the peer reviewed English language medical case literature and analyzed 324 cases that contained detailed individual case information. This search yielded 54 cases in which "some fashion of admission was noted that the injured baby had been shaken" (author's words). The details for all 54 cases were analyzed and of those, 11 cases were found wherein the reviewed material yielded no evidence of impact, and 12 cases had evidence of impact. The remaining cases provided either insufficient information or were excluded from the series for other reasons. After attempted statistical analysis of the material, no conclusions could be drawn by the author regarding which injuries occurred because of inflicted or accidental physical forces or by underlying or secondary disease processes, chiefly due to a paucity of data and inconsistent recording of relevant clinical information. That is, it was impossible to determine with scientific rigor what role shaking may have played in abusive head injury in these reported cases. Finally, it was not possible from the case analyses to infer that any particular form of intracranial or intraocular pathology was causally related to shaking, and that most of the pathologies in allegedly shaken babies were due to impact injuries to the head and body.

Science and Shaking

The practice of clinical medicine is considered to be an artistic and a scientific endeavor. In its highest form this is accomplished through the elicitation of a careful and accurate history, and the performance of a thorough physical examination. It is in this manner that an appropriate diagnosis or differential diagnosis is made. In doing so, the physician must understand the principles underlying the normal and pathologic characteristics of the individual patient before him, and how to identify accurately, delineate, and ultimately integrate these characteristics in a way that elucidates the condition of his patient clearly and concisely. The understanding of such principles, however intricate and a priori compassionate, must be grounded in objectivity, logic, and rationality, and must be in conformity with known biologic and physical laws.

There is a balance between the qualitative aspect of caring for people who are sick, and the quantitative, ultimately cognitive understanding of science underlying the practice of clinical medicine. Although this latter understanding may be considered an applied science, it is grounded in principles of science nonetheless. Thus, within the framework of our approach to medicine, the same principles of scientific methodology and understanding are relevant as they are to understanding the nature of the world around us.

Advances in such scientific understanding may occur in two different ways. The first is by objective observation of phenomena occurring in nature, and correlation of this observation with what is already known of the physical universe to produce a more complete understanding and a higher order of comprehension. The second method is by experimentation under controlled conditions where investigators test hypotheses formulated in a methodical, logical, and rational way, in order to explain observed phenomena. This is how our understanding of the world advances, and this is also precisely how medicine advances. Verification of observation leads to verification by experimentation. This is sound scientific methodology. When this methodology produces descriptions and explanations that are in conformity, one has glimpsed a truth. When such descriptions and explanations are at variance, something is amiss, and truth is not identified.

References

1) Bandak FA: Shaken baby syndrome: a biomechanics analysis of injury mechanisms. *Forensic Sci Int* 151: 71–79, 2005
2) Caffey J: On the theory and practice of shaking infants. Its potential residual effects of permanent brain damage and mental retardation. *Am J Dis Child* 124: 161–169, 1972
3) Caffey J: The whiplash shaken infant syndrome: Manual shaking by the extremities with whiplash-induced intracranial and intraocular bleeding, linked with residual permanent brain damage and mental retardation. *Pediatrics* 54: 396–403, 1974
4) The Child Abuse Prevention and Treatment Act (CAPTA) of 1974, Public Law No 93-247 (January 31, 1974)
5) Cunningham F, Hauth J, Leveno K, Gilstrap L III, Bloom S, Wenstrom K: *Williams Obstetrics, ed 22.* New York, McGraw-Hill Medical Publishing Division, 2005, section V: p 682
6) Donohoe M: Evidence-based medicine and shaken baby syndrome: part I: literature review, 1966–1998. *Am J Forensic Med Pathol* 24(3): 239–242, 2003
7) Duhaime A, Gennarelli T, Thibault L, Bruce D, Margulies S, Wiser R: The shaken baby syndrome. A clinical, pathological, and biomechanical study. *J Neurosurg* 66: 409–415, 1987
8) Guthkelch AN: Infantile subdural haematoma and its relationship to whiplash injuries. *Br Med J* 2(759): 430–431, 1971
9) Holbourn AH: Mechanics of head injuries. *Lancet* 9: 438–441, 1943
10) Holbourn AH: The mechanics of trauma with special reference to herniation of cerebral tissue. *J Neurosurg* 1: 191–200, 1944
11) Ito H, Komai T, Yamamoto S: Fibrinolytic enzyme in the lining walls of chronic subdural hematoma. *J Neurosurg* 48: 197–200, 1978
12) Ito H, Yamamoto S, Komai T, Mizukoshi H: Role of local hyperfibrinolysis in the etiology of chronic subdural hematoma. *J Neurosurg* 45: 26–31, 1976
13) Kawakami K, Chikama M, Tamiya T, Shimamura Y: Coagulation and fibrinolysis in chronic subdural hematoma. *Neurosurgery* 25: 25–29, 1989
14) Leestma JE: Case analysis of brain-injured admittedly shaken infants: 54 cases, 1969–2001. *Am J Forensic Med Pathol* 26(3): 199–212, 2005
15) Merriam-Webster Online Dictionary. Springfield, Mass, Merriam-Webster, Inc, ©2005. Odyssey (noun). Available from: http://www.m-w.com/dictionary.htm
16) Ommaya AK, Faas F, Yarnell P: Whiplash injury and brain damage. *JAMA* 204: 75–79, 1968
17) Prange M, Coats B, Duhaime A, Margulies S: Anthropomorphic simulations of falls, shakes, and inflicted impacts in infants. *J Neurosurg* 99: 143–150, 2003
18) Weber W: [Experimental studies of skull fractures in infants]. *Z Rechtsmed* 92: 87–94, 1984 (Ger, with Eng abstract)
19) Weber W: [Biomechanical fragility of the infant skull]. Z Rechtsmed 94: 93–101, 1985 (Ger, with Eng abstract)
20) Weber W: [Predilection sites of infantile skull fractures following blunt force]. *Z Rechtsmed* 98: 81–93, 1987 (Ger, with Eng abstract)
21) Yamashima T, Yamamoto S, Friede R: The role of endothelial gap junctions in the enlargement of chronic subdural hematomas. *J Neurosurg* 59: 298–303, 1983

Address reprint requests to: R. H. Uscinski, M.D., Potomac Institute for Policy Studies, Ballston Metro Center Office Tower, 901 Stuart St. #200, Arlington, VA 22203, U.S.A.
e-mail: ruscinski@potomacinstitute.org

EXHIBIT P

STATE OF _____)

)

COUNTY/CITY OF _____)

AFFIDAVIT OF DR. RONALD USCINSKI

I, Dr. Ronald Uscinski, make the following statement under penalty of perjury:

1. I am a neurosurgeon who testified as an expert witness on behalf of Trudy Muñoz Rueda in January of 2010. My resume is attached.

2. Guillermo Uriarte contacted me and asked me to be an expert witness in the case of *Commonwealth v. Trudy Munoz Rueda*. Although Mr. Uriarte eventually gave me the materials I needed, there was confusion preparing for the trial because it appeared the attorneys were insufficiently prepared. There was a large volume of material and it takes a considerable amount of time for defense attorneys to absorb the material and become familiar with the relevant facts and concepts in alleged shaken baby syndrome ("SBS") cases. Simply put, there is a significant learning curve. In this case, it was clear that the attorneys lacked a solid foundation in the medical records and the issues. Moreover, my recollection is that the defense attorneys did not provide the medical records to me until approximately a few weeks before trial. This late disclosure prevented a thorough enough review of the records before trial, and the attorneys did not call my attention to any relevant information in them.

3. I have testified in over 160 SBS cases, and Trudy's defense attorneys did not seem adequately prepared. My experience has been that experts do not win cases; lawyers win cases. In an alleged SBS case like Trudy's – where acute trauma has been presumed in spite of the absence of medical evidence supporting such a conclusion – it is particularly important for the defense attorneys to be well-versed in both the competing medical theories at issue and the facts of the case. In this case, the defense had to demonstrate three primary issues for the jury: (1) that trauma is not the only etiologic explanation for subdural hematoma and retinal hemorrhages; and (2) that there were several other possible and non-traumatic or certainly nonacute traumatic medical causes of Noah Whitmer's condition on April 20, 2009 and (3) the scientific foundation for so-called "Shaken Baby Syndrome" is to this date lacking.

4. I testified in a case in Fairfax County several months after this case that had many of the same elements and treating physicians and expert witnesses. The primary difference was that there was a well-prepared public defender in the other case, Dawn Butorac, who was well-versed in the facts of her case, the central issues in SBS cases, and the competing theories about the causes. The defendant in the other case was acquitted. The attorneys in this case may have been excellent attorneys in their areas of expertise, but they appeared to have insufficient background for successful defense here.

5. I routinely testify for the defense in SBS cases for two reasons; First, it seems SBS remains after more than 40 years an unproved, in fact scientifically nonplausible hypothesis that is used to prosecute people in cases with scant or no correlating physical evidence. "Shaken Baby Syndrome" is a construct that allows doctors and medical personnel to assert, without scientific proof, that if an infant or a child has a subdural hematoma and retinal hemorrhaging, the presumption of violent manual shaking must be made. Ms. Munoz' case is a prime example of how unproven SBS theories can lead to the conviction of innocent caretakers. Second, I have yet to be contacted by any prosecuting attorney to review such a case.

6. Once INOVA-Fairfax medical personnel saw scans that revealed a subdural hematoma and retinal hemorrhaging, they presumed Noah Whitmer was abused and did not seem to consider alternative explanations, even though there were additional findings in this case that warranted further explanation and an expanded differential diagnosis.

7. In this case, the treating physicians and medical personnel ignored or overlooked several potentially relevant facts in Noah's Whitmer's records. Upon admission to INOVA Fairfax Hospital, Noah Whitmer's father reported that Noah had a cough, that he developed a fever, that sputum tests showed the presence of pneumonia and strep bacteria, that his chest x-ray revealed indications of respiratory infection, and that the hospital treated these with a course of antibiotics. Infection is a common trigger for venous thrombosis, as the Commonwealth's witnesses acknowledge.

8. It also appears that INOVA's doctors never tested Noah for meningitis, even though Dr. Handa at the Wilmer Eye Institute noted that, other than a bleeding diathesis or leukemia, meningitis was the only real other diagnosis that would have the same distribution of retinal hemorrhages as "shaken baby syndrome."

9. Medical personnel at INOVA–Fairfax also appear to have failed to conduct a thorough and meaningful medical history, beyond making note of three potentially relevant facts reported by Michael Whitmer, Noah's father, on the evening of Noah's admission to the hospital in critical condition. Michael Whitmer told medical personnel that Noah's paternal grandfather had a history of febrile seizures, and that he himself had two male cousins with

muscular dystrophy. Michael Whitmer told medical personnel that Noah's mother had a maternal relative who died at age 8 of some "chromosomal abnormality." There is no indication in the records that medical personnel pursued any one of these potentially relevant issues any further.

10. Noah's parents also reported that a wooden plaque had fallen on Noah's head approximately ten days before his admission to the hospital. No further consideration of this fact is indicated in the medical records.

11. INOVA-Fairfax lab reports reflect that in the PICU unit, sputum culture showed Staph Aureus and Strep pneumonia, and antibiotics were prescribed. Infection is a known cause of venous thrombosis, and Noah Whitmer clearly had an infection.

12. The defense attorneys appeared to be struggling to prepare adequately before trial. A large volume of medical records were provided without calling attention to signs of infection, a history of febrile seizures, muscular dystrophy, chromosomal abnormality, or a previous head trauma. Further, it is now known that multiple individuals with whom Noah spent a great deal of time in the week preceding the onset of his symptoms reported that he was acting fussy and cranky. Issues such as irritability or dehydration while nonspecific, could represent the prodrome of such entities as infection and/or venous thrombosis, which was seen in Noah's imaging and is one potential cause of his symptoms. Even for a neurosurgeon, evidence of infection, a family history that includes chromosomal abnormality or unusual inflammatory response is relevant indication of a different etiology than SBS.

13. In a case such as this, where CT and MRI scans were central to both the Commonwealth's case and to a valid defense, the assistance of a neuroradiologist such as Dr. Patrick Barnes was essential. As a neurosurgeon who routinely reviews scans, although my testimony would be important with regards to anatomy and particularly traumatic anatomy, as well as imaging relevant to surgical planning and execution, a neuroradiologist would be helpful and at times critical in adding the further dimension of nonsurgical findings and interpretations as further evidence of nontraumatic (such as infectious and vascular) etiologies other than SBS, including cortical or sinovenous thrombosis, the latter especially when aggravated by such factors as infection or dehydration. I do not know why Dr. Barnes did not testify at trial.

14. A normal, vaginal birth can and indeed does cause head trauma to the infant being born. In a recent study of 101 live births, subdural hematomas were discovered in 46 of those infants. Most of the time, these resolve naturally and without issue for the infant. But occasionally, if the normal healing processes fail and the subdural blood is not reabsorbed, the hematoma enlarges slowly over time by rebleeding into the subdural space with

accompanying compensatory enlargement of the infant's head. The presence of evolving chronic subdural collections of blood occurring during birth trauma are thus essentially pre-existing conditions. In my opinion, this series of events is yet another likely medical explanation for Noah Whitmer's symptoms. Testimony from Dr. Barnes therefore might have differed from yet at the same time synergized with the evidence I identified in this case suggesting that Noah Whitmer suffered a re-bleed of a chronic subdural hematoma.

15. It is also possible that the lengthy efforts to resuscitate Noah Whitmer caused the retinal hemorrhages. For forty-five minutes, this 4 month-old child was receiving forced air and chest compressions. Meningitis, which is inflammation of the meninges, or coverings around the brain, could also cause retinal hemorrhages.

16. I was prepared for this case the weekend before the trial by Jim Kearny. The defense team did not seem to have formulated a cohesive strategy for presenting the expert testimony, it appeared they were still endeavoring to understand the medical records and issues.

17. I understand that signing an affidavit is similar to testifying in Court. I have carefully reviewed this affidavit before signing it to ensure that it is accurate to the best of my recollection.

FURTHER AFFIANT SAYETH NAUGHT.

Ronald Uscinski

Signed and sworn before me this _____ day of _____ 2012.

_____
NOTARY PUBLIC

My commission expires: _____
Notary registration no.: _____

RONALD H. USCINSKI, M.D.
NEUROLOGICAL SURGERY

5530 Wisconsin Ave, #1147
Chevy Chase, Maryland 20815
Tel. 301-656-8590
Fax. 301-656-8593

CURRICULUM VITAE

Ronald H. Uscinski. M.D., FACS

Occupation: Physician

Education:

B.S., Fordham University, New York, NY, 1964

M.D., Georgetown University, Washington, D.C., 1968

Internship, Bronx Municipal Hospital Center, Albert Einstein University College of Medicine, New York, NY, 1968-9

Residency in Neurological Surgery, Georgetown University and affiliated Hospitals, 1971-1975

Military Experience:

Medical Officer, United States Navy; served with United States Marine Corps, Parris island, South Carolina, and aboard The U. S. S. Thomas A Edison (SSBN 610-B) Atlantic Submarine Force, 1969-1971

Appointments & Positions:

Senior Surgeon, U.S. Public Health Service, Medical Officer, Surgical Neurology Branch, National Institute of Neurological and Communicative Disorders and Stroke, NIH, Bethesda, Maryland, 1975-1976

Instructor in Surgery (neurosurgery) Georgetown University School of Medicine, Washington D.C., 1975-1976

Consultant in Neurosurgery, NIH, Bethesda, Maryland, 1976-1977

Clinical Instructor in Neurosurgery, Medical University of South Carolina, Charleston, South Carolina, 1977-1980

Clinical Assistant Professor, Dept. of Surgery (Neurosurgery), Georgetown University School of Medicine, Washington D.C., 1980-2000

Clinical Associate Professor, 2000-present

Clinical Assistant Professor, Department of Pediatrics, Georgetown University School of Medicine, Washington D.C., 1980-present.

Clinical Assistant Professor, Department of Neurological Surgery, the George Washington University School of Medicine, 1997-2008.

Clinical Associate Professor, 2008-

Adjunct Research Fellow: Potomac Institute for Policy Studies, Arlington, Va, 2004-2006.
Senior Adjunct Fellow, 2006-

Certification

American Board of Neurological Surgery, 1978

Societies:

Congress of Neurological Surgeons, 1975
American Medical Association 1976
South Carolina Medical Society, 1977-1980
American Association of Neurological Surgeons, 1979
American College of Surgeons, 1980
District of Columbia Medical Society, 1981
Polish Society of Neurological Surgeons, corresponding member, 1983
Research Society of Neurological Surgeons, 1989

EXHIBIT Q